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Archives of Neurology and Psychiatry

Vol. 2 DECEMBER, 1919 No. 6

ACUTE ASCENDING PARALYSIS AMONG TROOPS

PATHOLOGIC FINDINGS *

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NEW YORK

Among the hitherto little known diseases that appeared among the troops in France was a nervous affection of considerable rarity, but of such uniformity in symptomatology and general course that it easily became evident that this was a true disease entity. Gordon Holmes reported a series of cases of this condition in 1917, and later Bradford, Bashford and Wilson,[1] in an excellent article, reported at considerable length on the disease from observations on a series of thirty cases. The comparative rarity of the condition may be judged from this number as it represented all they could assemble from the large number of British troops in France, and it probably represents a goodly proportion of all the cases that occurred.

The writer saw four or five of these cases in eleven months' service with the British Expeditionary Forces, and in nine months' service with the American troops in France, but one case came under his observation. I cannot but feel that this does not represent a fair proportion of the cases that occurred among our troops. Not a single case was seen by me in the big hospital group at Bazoilles, where seven base hospitals had a total of nearly 20,000 beds. The one American patient reported in this article was seen by me in a field hospital to which he had been sent with the diagnosis of psychoneurosis. The divisional psychiatrist, Major George A. Blakeslee, recognizing that the disease was unfamiliar to him, asked to have the writer see the patient. Obviously, the condition was not recognized up the line as being of an organic nature and very likely a number of other similar cases may have slipped through on a similar diagnosis. Lieut.-Col. Sanger Brown, 3rd, told me when I spoke to him of the disease that he was certain

* Read before the Forty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 16-18, 1919.

1. Bradford, Bashford and Wilson: Acute Infective Polyneuritis, Quart. J. Med. **12**:88 (Oct.) 1918; (Jan.) 1919.

he had seen one case of it at Base Hospital No. 8 at Savanay which had slipped through undiagnosed.

Bradford's careful description of the clinical phenomena of the disease corresponds exactly with that of all the cases observed by the writer. A short fever of from 100 to 103 F., lasting from two to four days, accompanied by severe headache and general pains, subsides and leaves the patient feeling comfortable enough to continue his usually quite arduous duties in the line. In the latent period that now intervenes no symptoms are to be observed for a period of four or five days to a month or six weeks. Following this latent period, the paralytic stage makes its appearance usually quite suddenly. As a rule, the patient suddenly finds his legs getting weak so that he is obliged to sit down. After resting a while he tries to arise and finds that he is unable to do so. When he is removed to the hospital it is found that both legs are completely paralyzed. This paralysis is of an extreme flaccid type, all tendon reflexes being absolutely abolished. At this time the patient is usually found to have a slight fever (99-101 F.),

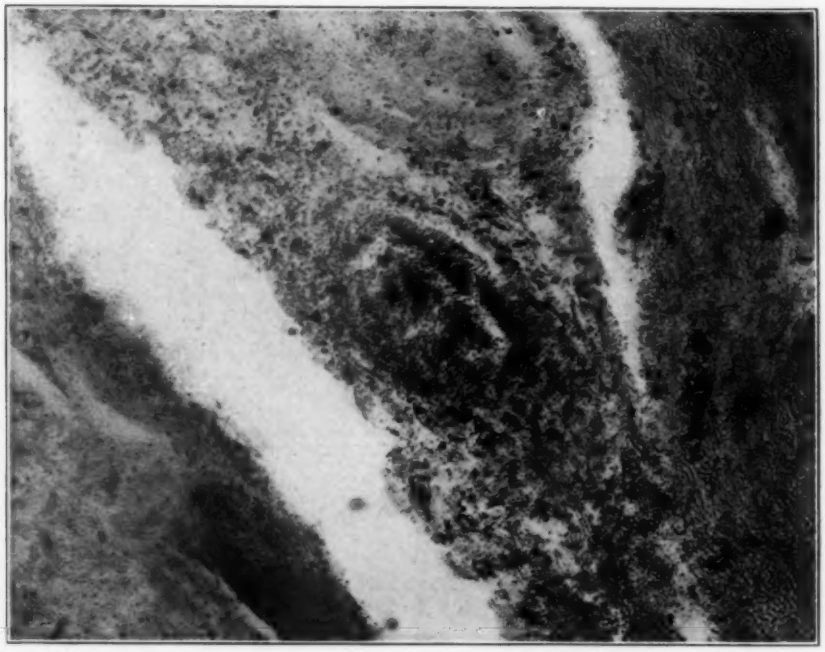

Fig. 1.—The membranes of the cord, low power. Case 1: At the lower left hand corner is the surface of the cord covered by thickened pia. To the right of this is the arachnoid, much thickened, hyperemic, with the tissue spaces filled with red blood cells and detritus. At the right edge of the illustration is the dura, which is adherent to the arachnoid at the lower part.

and frequently complains of pain in the back. The paralysis usually progresses in an ascending fashion. The trunk musculature becomes progressively involved, and after from about 24 to 48 hours a paralysis, similar to that in the legs, is seen in the arms. Following this, the phrenic and vagus nerves may be affected and the patient dies of respiratory paralysis. If this does not occur the paralysis may ascend so far as to involve both facial nerves. Bradford has seen but one case in which the sixth nerve was involved, but never any of those located more cephalad.

When the paralytic stage is fully developed, the patient is practically completely paralyzed from the level of the seventh nerve down. If

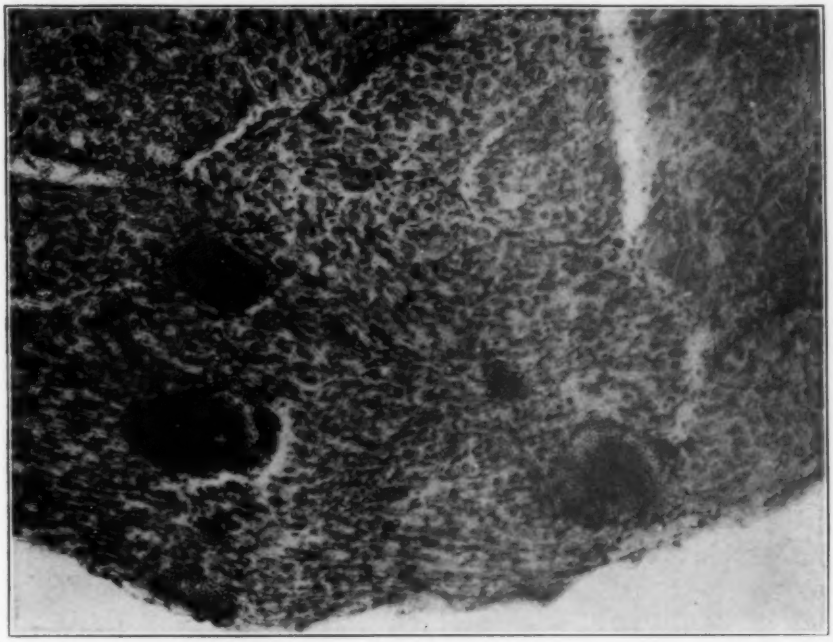

Fig. 2.—Anterior nerve root in the arachnoid. Case 1: showing marked hyperemia and hemorrhage.

any part of the somatic musculature is spared, it is usually the distal parts of the limb. Thus a patient may be able to move his hands fairly well long after motor power is lost at the elbow and shoulder. The paralysis is always of the flaccid type with complete loss of the tendon and superficial reflexes. Sensory loss is always much less marked than motor. In early cases no sensory involvement can be determined. Later in the disease there is present a glove and stocking shaped anesthesia of much the same type as is seen in multiple neuritis. The sensory signs, like the motor, are always bilateral. The prepon-

derance of motor over sensory manifestations is important in view of the pathologic findings. The sphincters are rarely, if ever, affected. The blood may show a moderate leukocytosis. The spinal fluid is always completely negative.

The mortality is fairly high. In Bradford's thirty cases, death occurred in eight, half of the deaths occurring within one week of the onset of the palsy. In the nonfatal cases the paralysis remains stationery for a while, and then starts slowly to improve. Usually the face first shows improvement, and then slowly the arms and legs get stronger, and after from six to eight months the patient has recovered without residual signs of the disease.

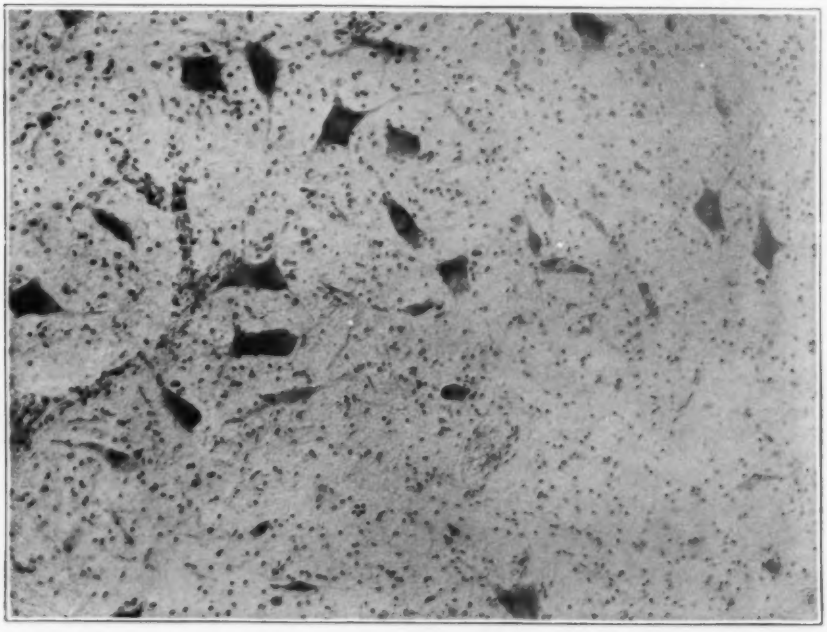

Fig. 3.—Anterior horn, low power. Case 1: showing especially the increase of the neuroglia cells.

PATHOLOGY

Bradford in the article mentioned in the foregoing, gives his results from a study of the postmortem material from six cases. Unfortunately, he makes very little mention of the condition of the meninges in which the most striking changes were seen in my cases. In one case only does he mention the membranes (Case 2), and then observed macroscopically "great edema of the pia-arachnoid and marked distention of the theca." In the spinal cord itself he noted a number of changes in the nerve cells, especially those of the anterior horn. In

some of his cases these cells were more severally affected than in others. In the more advanced cases the motor cells showed eccentricity of the nucleus, finely divided tigroid substance, which was grouped around the periphery of some cells and gathered in the center of others. These, which he shows in excellent illustration, present quite the typical central chromatolysis of secondary degeneration. Many of the changed cells that he describes and pictures, especially the shrunken ones with crenated outlines, appear more typical of post-mortem changes than any known type of cell degeneration. Likewise, I am inclined to doubt the pathognomonic significance of the deep diffuse staining of certain tract cells described by Bashford, for such cells

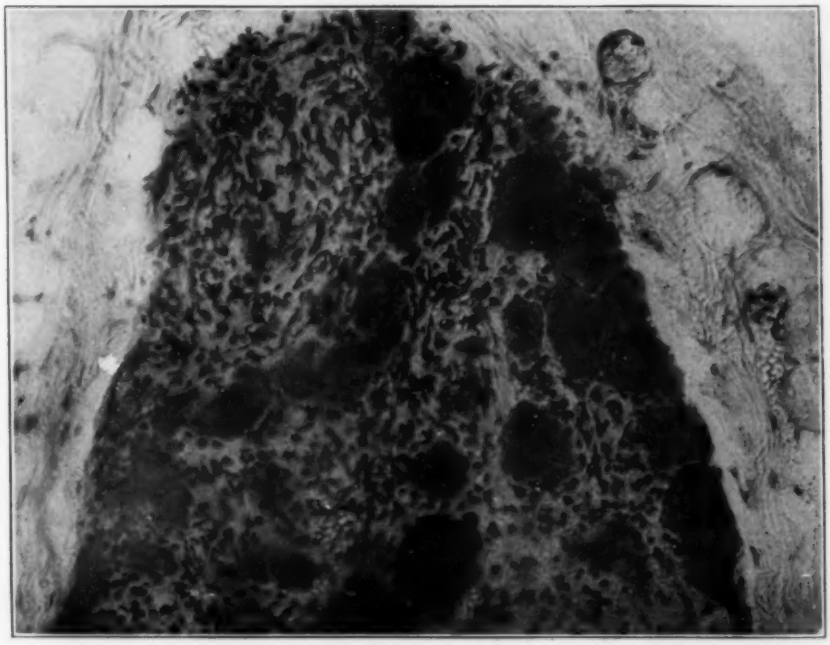

Fig. 4.—Posterior nerve root ganglion. Case 1: showing the increase of interstitial cells and diffuse staining of the nerve cells.

are frequently seen in control cords where no nervous disease had existed. Axis cylinders sometimes appear swollen. He describes some cells in which the outlines were definitely hollowed out where a round cell was applied. This is certainly neuronophagy and must be considered a true part of the pathologic picture.

Besides the changes in the nerve cells, Bashford describes a marked increase in the number of "small round cells" in the gray matter of the cord. This he describes "a round cell infiltration," but he makes a sharp distinction between this and the infiltration of the vessel

sheaths with lymphocytes seen in poliomyelitis. From Bashford's description and pictures, as well as from the study of my own sections, it is evident that these cells are small, young neuroglia cells, and not cells of inflammatory mesodermal origin. In the white matter of the cord, Bashford describes little that is pathologic except evidences of Wallerian degeneration in some of the fibers and an increased number of small, round cells (neuroglia) in the region of the anterior and posterior roots.

The posterior root ganglions of four of Bashford's cases appeared negative except for some increase in the number of small round cells.

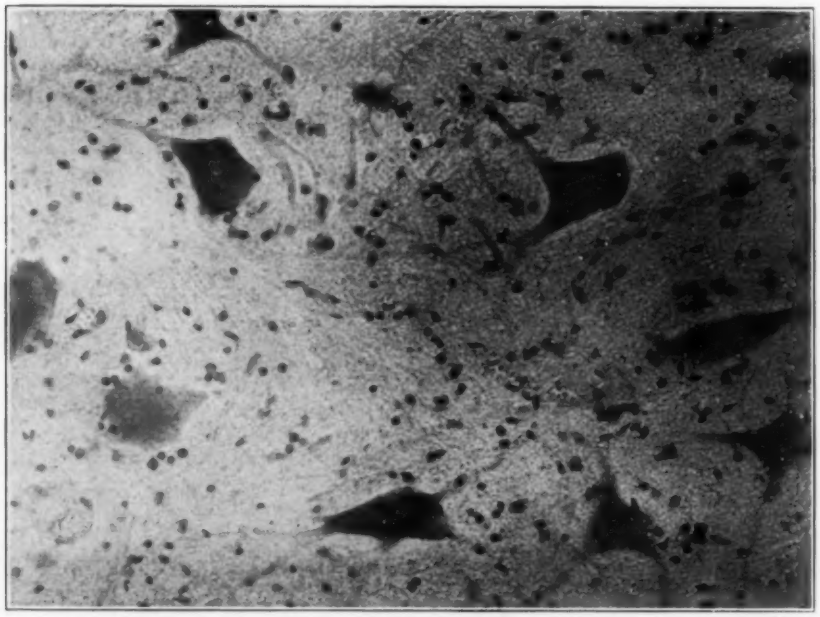

Fig. 5.—Anterior horn, high power. Case 1: showing increase of neuroglia and diffuse staining of nerve cells (acute cloudy swelling).

In one case the cervical glanglions appeared normal, while those from the lumbar region were markedly altered. These showed eccentricity of the nucleus, disappearance of the tigroid and vacuolation. The number of "round cells" was increased.

The peripheral nerves examined showed similar features of varying intensity in different parts of the same nerve. In some cases a partial Wallerian secondary degeneration was seen, and in others an active neuritis with hyperplasia of the Schwann cells, inflammatory exudate of "round cells," and hemorrhages. Sometimes the medullary

sheaths were quite swollen and the axis cylinders were sometimes swollen, irregularly stained and fragmented.

The cerebral cortex and the cerebellum showed the same increase of neuroglia cells as was observed in the cord.

Muscles showed signs of fatty degeneration with osmic and Marchi stains.

Bashford was able to produce the disease in monkeys (Macaccus rhesus) by intradural injection of a glycerin emulsion of the spinal cord of fatal human cases. The clinical features and pathologic findings in these monkeys were the same as in man. Likewise the disease was similarly transmitted from monkey to monkey.

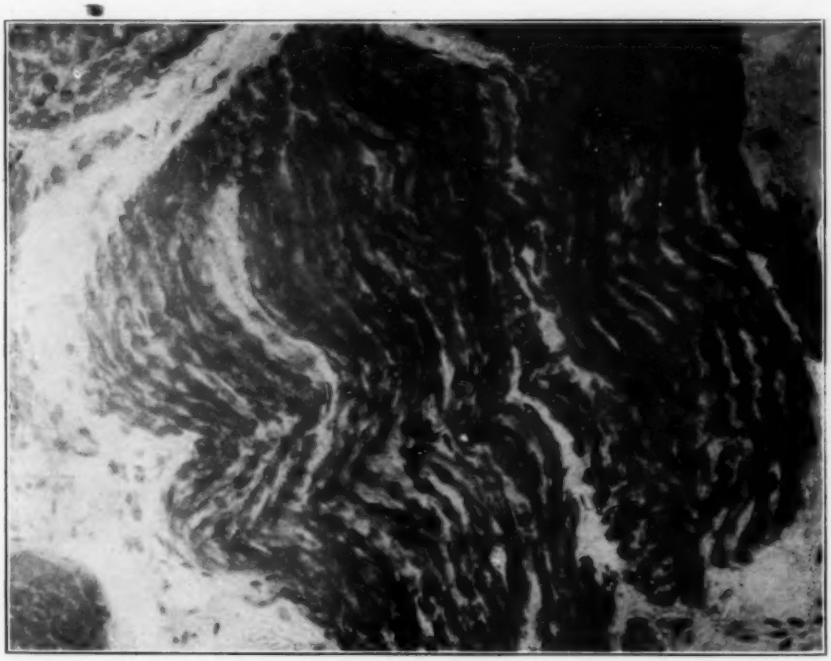

Fig. 6.—Anterior nerve roots in the arachnoid, high power. Case 1: showing the beady appearance of the axons in acute degeneration.

Wilson has made a careful study of the bacteriology of the disease and succeeded in isolating an organism in pure culture that produced the disease in monkeys and which was recovered in pure culture from the inoculated monkey after death. He describes the organisms as being "minute rounded, oval or kidney-shaped bodies, measuring 0.2 to 0.5μ in diameter, occurring in groups, more rarely in pairs or short chains." In young cultures it appears as a darkly stained spot, eccentrically placed, surrounded by a narrow faintly stained area. In

older cultures the organism swells and loses its selective staining. It is anaerobic and grows only on Flexner and Noguchi's tissue agar-serum bouillon medium.

AUTHOR'S CASES

The material for this paper consists of the spinal cords from two cases. Only the first case was seen by the author before death. Neither history is available at present. The history and other records of Case 1 are in the possession of Major Blakeslee, who has not yet returned from France. The history of Case 2 was transmitted verbally

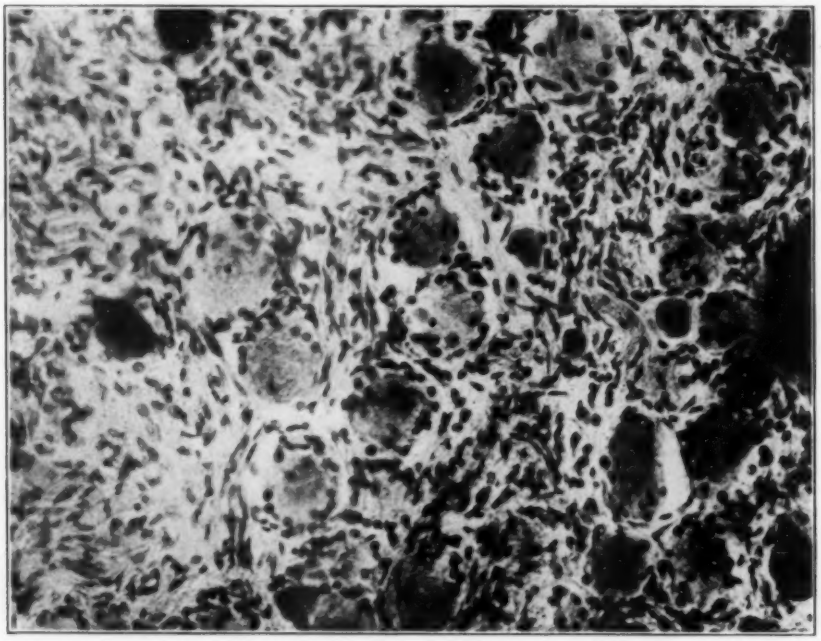

Fig. 7.—Posterior spinal root ganglion, high power. Case 2: showing faint diffuse staining of ganglion cells, increase of interstitial cells and marked neuronophagy.

in a very sketchy fashion by Major Young, Canadian Army M. C., of No. 4 Canadian Casualty Clearing Station, who observed the case. Hence the histories will be given very briefly from incomplete notes. In both cases the spinal cords together with posterior nerve root ganglions were preserved in liquor formaldehydi. Part of the material was later treated with Zenker's fluid. The blocks were embedded in celloidin and the section stained with hematoxylin-eosin, cresyl violet, iron hematoxylin and by the von Gieson, Mallory's anilin blue and Unna-Pappenheim methods.

CASE 1.—*History.*—Private, 320th Field Artillery, A. E. F., aged 42, admitted to the triage of the 82d Division on the night of Oct. 27, 1918, stated he had had a fever for about twenty-four hours, five days before the onset of the paralysis. After this he was well until October 26, when he suddenly became weak in the knees while working with a pick. He went to his dugout and lay down, and after a few hours was unable to move his legs. He was kept in his dugout, and the next morning found he could not move either legs or arms. He was then sent to the triage and seen by the author on the morning of the twenty-eighth. At this time he was completely paralyzed in the legs and trunk, but was not incontinent of feces or urine. His arms were completely paralyzed with the exception of the fingers of the right hand, which he could move voluntarily to a slight extent. His pulse was rapid

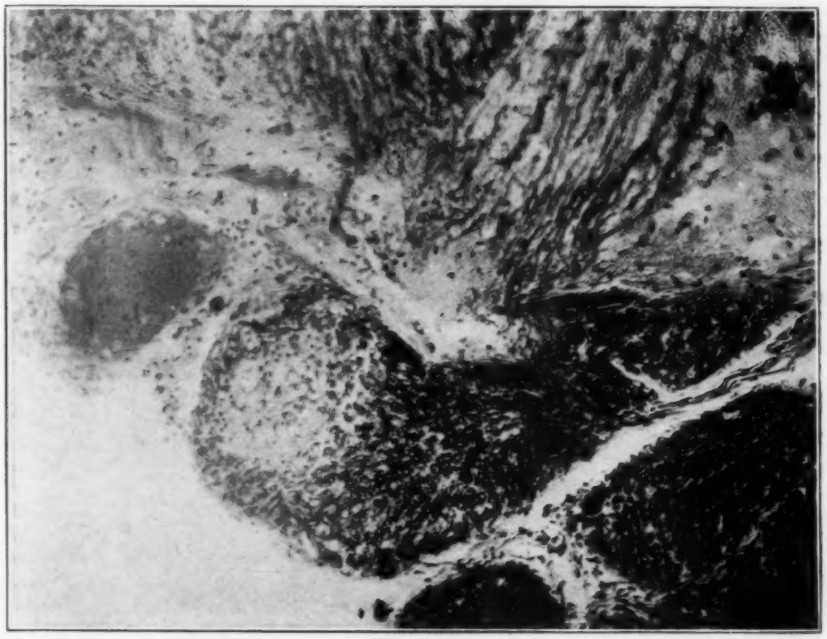

Fig. 8.—Exit zone of anterior root fibers, low power. Case 2: showing degeneration in the emerging fibers and a patch of severe degeneration in the root just outside the cord.

and he had a temperature of 101 F. His breathing was labored and he had a beginning edema of the lungs. He appeared acutely ill. The paralysis was of an extreme flaccid type with complete abolition of all reflexes. No sensory loss could be determined anywhere. He died at 7:30 a. m., October 29, less than three days after the onset of the paralysis. The necropsy was performed by Major Blakeslee under the inconvenient condition of active service in the midst of the strenuous campaign in the Argonne, but the cord was carefully removed with all the membranes and posterior root ganglions intact and well preserved in liquor formaldehydi.

Histologic Findings.—Mesodermal Elements: The dura appeared normal throughout. In some places in the epidural fat the blood vessels were dilated

and packed with red blood cells (active hyperemia). Around these dilated vessels there was to be seen considerable free-lying blood, but it is uncertain that this was not a postmortem phenomenon occurring at the time the cord was removed.

Quite the most striking change was seen in the arachnoid. This membrane, usually quite thin and difficult to demonstrate, was markedly thickened. This thickening consisted of marked hyperemia of the small blood vessels, edema of considerable degree, hemorrhage into the tissue spaces, thickening of the connective tissue fibers, formation of new fibers and young connective tissue cells and an amorphous stuff that appeared to be cell detritus (Fig. 1). In none of the sections was there an infiltration of other blood elements, except the red blood cells. In no place were there to be seen the layers and

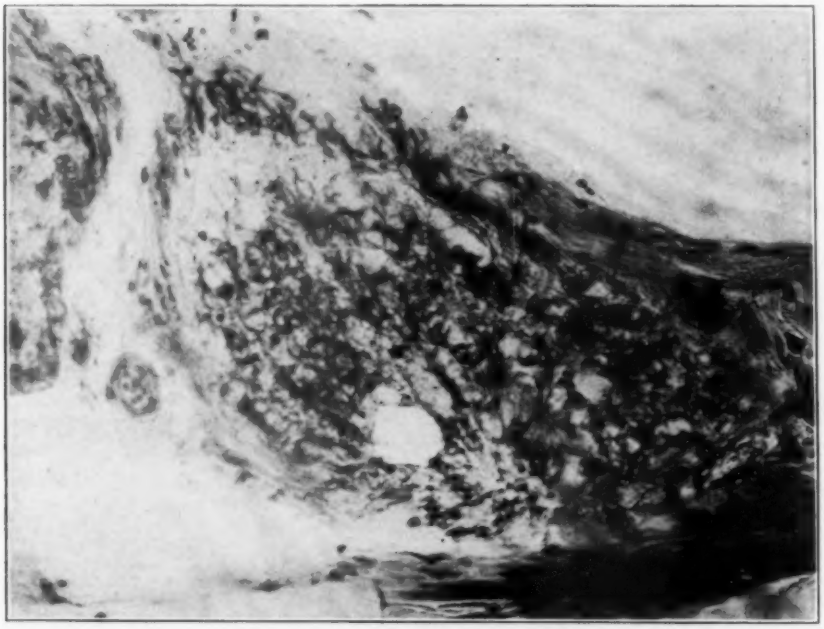

Fig. 9.—Cross section, anterior root just after emergence from the cord, high power. Case 2: showing complete degeneration of all the fibers.

clumps of polynuclear leukocytes as in septic meningitis, nor the lymphocytes and other related cells that form so prominent a part of the picture in tubercular meningitis, syphilis and poliomyelitis. This thickening of the arachnoid is always more marked over the anterior and anterolateral aspects of the cord than over the posterior. Often it is marked around the nerve roots that lie in the arachnoid and especially those around the anterior roots. In places the process has progressed so far that the arachnoid is so closely adherent to the pia that no line of separation can be determined and, more rarely, all three membranes appear to have fused into one, as though the inflammatory arachnoid had acted as cementing structure between the pia and dura.

The pia mater appeared rather thickened, mostly from swelling of the connective tisue fibers and partly by adhesion to it of the arachnoid. The

blood vessels revealed moderate hyperemia, but the tissue spaces were not enlarged nor was there any infiltration of these spaces with lymphocytes. The pial involvement was usually most marked over the anterior surface of the cord.

The condition of the blood vessels of the meninges has already been described. One notes only hyperemia with no change in the vessel walls and no perivascular infiltration. In the spinal cord itself the same conditions obtained. Throughout there was marked hyperemia, especially in the central gray. The vessel walls appeared normal and in no instance showed the ring of lymphocytes lying in the Virchow-Robin space, which is so char-

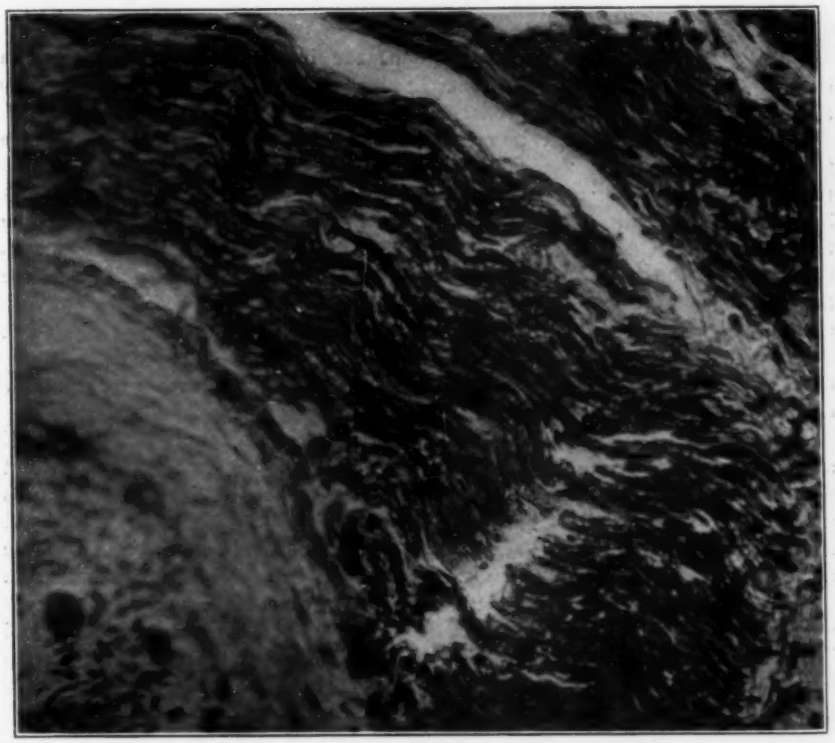

Fig. 10.—Posterior nerve root fibers in the neighborhood of the ganglion. Case 2, showing beginning degeneration of the fibers and "neuro-keratin network."

acteristic of anterior poliomyelitis. The artificial perivascular spaces were widened in some spots, but never contained degenerating cells. In a few places red blood cells might be seen lying outside of vessels, sometimes around vessels, but practically never to such an extent as to indicate active hemorrhage. As Bashford has stated, this may be a postmortem phenomenon. In the nerve roots of the arachnoid and especially around the anterior roots, the hyperemia was still more marked than in the cord and here larger hemorrhages might be seen which may well have occurred antemortem (Fig. 2).

The Ectodermal Elements: The superficial layer of neuroglia lying just under the pia showed no changes. All neuroglia fiber, in so far as they were brought out with the stains, appeared normal in appearance and number. The cellular neuroglia showed considerable change. In the central gray, especially in the anterior horns, the neuroglia cells were greatly increased in number. These new cells had the round or oval nuclei, either darkly stained or showing the definite reticulum and the small, at times invisible, cytoplasm of young glia cells. These are the cells that Bashford calls "small round cells" and to me they appear as being entirely of neuroglia origin. In some places they are rather more numerous around some anterior horn cells (Fig. 3). Neuronophagy, as described by Bashford, was not seen. In the white matter of the cord the neuroglia cells of the anterior and posterior roots, as Bashford described, were increased.

In the posterior spinal root ganglions the interstitial cells of neuroglia origin were likewise markedly increased in number and tended to group around certain of the nerve cells. No neuronophagy was seen (Fig. 4).

The Nerve Cells: The anterior horn cells showed but little change in this case. In most of the cells the tigroid substance appeared to be more finely divided than in normal cells, even for cells fixed in liquor formaldehydi. In some cells the nucleus was rather eccentrically placed, and the tigroid was lessened in amount, especially in the center. It is difficult to say whether this was central chromatolysis due to destruction of the nerve fiber or the result of direct toxic action on the cell itself. While the picture may be looked on as pathologic, it surely must be in a very early stage (Fig. 5). As in Bashford's cases, so in mine, some of the cells of the endogenous cord fibers appeared altered in a similar manner as the motor cells, but always to a lesser degree.

The sensory cells of the posterior spinal root ganglions showed little if any change. In some section many of the cells appeared to take cresyl-violet rather faintly and the tigroid was very finely divided, but such pictures may be obtained at times with normal cells fixed in liquor formaldehydi. At any rate, there were no evidences of a real cell destruction. Some of the nuclei were eccentric but hardly more so than is often seen in normal material.

The Nerve Fibers: For some reason no pieces of the peripheral nerves were removed at necropsy. However, the nerve changes which were observed in the nerve root fibers were so striking and corresponded so closely with the nerve changes described by Bashford that enough can be seen in them to account for the symptoms. The blood vessels of the nerve roots were all widely dilated in marked hyperemia and in a number of cases hemorrhages had occurred. Degeneration of varying degrees was the ruling picture. In many places the nerve fibers showed signs of degeneration where they passed through the pia and just inside the cord. In sections stained by the Marchi method was seen very marked degeneration in the posterior nerve root fibers where they run in the arachnoid and more especially just after they have entered the spinal cord. These fibers lay as a black band in the root entrance zone of the posterior columns, by far the most striking thing in the section. In every case the maximum degeneration was to be seen in the fibers just after they leave the cord and begin to be enveloped by the arachnoid. The degeneration was so marked here that it would appear that this was the point of most extensive involvement of the peripheral nerves. The type of degeneration at this point appeared decidedly toxic while in the fibers, both above and below this point, the degeneration appeared to be of the

Wallerian, secondary type. Where the nerves enter the arachnoid, the degeneration was usually pretty complete. Some swollen axis cylinders were to be seen, but most of them had disappeared. The myelin sheath was completely gone. Many of the Schwann cells disappeared and the connective tissue trabeculae appeared to be diminished. Further peripherad the degeneration was of more moderate degree. Here also, on longitudinal section the axis cylinders were swollen, knobby and broken. The myelin had broken up into large and small droplets. The neurolema was shrunken and the Schwann cells were much increased in number (Fig. 6). These changes are regularly more marked in the anterior roots and always very much slighter in the posterior roots. This corresponds very well with the clinical side of the picture in which the motor signs are always more marked than the sensory. Outside the dura the posterior roots near the ganglions show little, if any, alteration from the normal.

Summary of Histologic Findings.—The histologic findings were:

1. Hyperemia, hemorrhage edema, and fibrous swelling in the arachnoid; thickening of the pia.

2. No changes in blood vessel walls; no round cell infiltration.

3. Increase of the cellular neuroglia in the central gray, around the root fibers and in the posterior root ganglions.

4. Evidence of beginning degeneration of both a secondary and primary character in the anterior horn cells and some tract cells. Hyperemia of the central gray.

5. Marked degeneration of primary and secondary character of the nerve fibers where they lie in the arachnoid, always most marked in the motor fibers.

CASE 2.—*History.*—The history of this case is very incomplete, having been given to me from memory by Major Young, C. A. M. C., at a time when all the hospitals in Flanders were extremely busy with the wounded from the Passchendale battle. The patient was admitted to No. 4 Canadian C. C. S. about the middle of August, 1917, with a flaccid paralysis of both legs. The next day both arms were similarly paralyzed and later both seventh nerves were involved. The patient died about a week after the onset of the paralysis. A necropsy was performed by Captain Stokes, R. A. M. C., pathologist to No. 10 C. C. S. at Remy Siding, who kindly gave me a very generous share of the spinal cord segments and posterior root ganglions.

Histologic Findings.—Mesodermal Elements: The dura mater appeared to be perfectly normal in the few pieces at my disposal.

As in Case 1, the arachnoid showed marked changes but they were less easy to demonstrate on account of poor preservation due to the fact that the cord was not removed with the dura intact. However, one can see, in places, the hyperemia, edema, hemorrhage, swelling of the connective tissue fibers and cell detritus described in the previous case.

The pia mater is edematous, hyperemic and thickened somewhat more so than in Case 1. There is no infiltration with lymphocytes or leukocytes anywhere.

The blood vessels show a hyperemia in the gray matter of the cord but to a much less extent than in Case 1. The vessel walls show no change, and there is no infiltration of the Virchow-Robin spaces with lymph or other cells.

The Ectodermal Elements.—The fibrous neuroglia appeared normal, but the cellular neuroglia was increased in the central gray matter and around

the nerve roots to the same extent as in Case 1. There was no true neuronophagy. In the posterior root ganglions the neuroglia cells were markedly increased in number and neuronophagy was seen in many places.

The Nerve Cells: Many of the anterior horn cells appear quite normal while others show fine granulations of the tigroid and probably beginning cloudy swelling. The tract cells, as the motor, show on the whole less change than do those of Case 1.

The sensory cells of the posterior ganglions show, however, much more marked changes. The cells appear swollen and stain very pale. The edges of many are indented and phagocytic glia cells are to be seen invading the cytoplasm (true neuronophagy). Some of the cells stain diffusely dark and also show neuronophagy (Fig. 7).

The peripheral nerve fibers of the roots show even more degeneration than in Case 1, always more marked in the anterior roots. Just after these fibers emerge through the pia they often show a very extreme degeneration of a primary type (Figs. 8 and 9). Further peripherad in the arachnoid extreme degeneration is seen in longitudinal section—broken, swollen, knobby axis cylinders, disappearance of myelin, increase of Schwann cells and shrunken neurolemma. The extra dural portion of the posterior roots near the ganglions show a very interesting picture of moderately advanced degeneration. The axis cylinders appear rather normal. The myelin substance has broken up into large and small droplets still lying in the Schwann cells. The cytoplasm of the Schwann cells has apparently increased somewhat in amount and takes the stain darkly, showing a definite network between the fat droplets, giving a mosaic like appearance to the whole. This is the so-called "neuro-keratin network" described in normal and beginning degenerating nerves (Fig. 10).

Summary of Histologic Findings.—The histologic findings were:

1. Hyperemia, hemorrhage, edema, swelling in the arachnoid; thickening of the pia.
2. No changes in the blood vessel walls; no round cell infiltration.
3. Increase of the cellular neuroglia in the central gray, around the nerve roots and in the posterior root ganglions.
4. Slight secondary and primary degeneration in the anterior horn and a few tract cells; slight hyperemia of the central gray.
5. Marked primary and secondary degenerations in the anterior nerve root fibers, just as they emerge from the pia and where they run in the arachnoid.
6. Beginning degeneration in the posterior nerve roots just outside the dura.
7. Marked degeneration of the posterior root ganglion cells with neuronophagy.

PATHOGENESIS

It is quite obvious that the cases reported here and in previous articles represent a true disease of the nervous system which, until the publication of the article of Bradford, Bashford and Wilson, had never been accurately described. The way it affects the nervous system appears to be quite different from that of other acute diseases. The process is diffuse, although the peripheral system is always more severely damaged than the central. In the vertebral canal, not only the spinal cord, but also its membranes participate in the process. It is doubtful how the organism and its products reach the nervous system

where the changes occur, and from which the organisms have been recovered. The fact that the nerves are more severely affected at the arachnoid level than more peripherally would seem to indicate that the path of infection is not along the nerves.

The portal of entry must be by the blood or lymph channels. It is difficult to choose between these two, principally because of the fact that there is no lymphocytic proliferation. What evidence there is points rather to the lymphatic conveyance. Bashford states that in his monkey cases "the only constant features — outside of the nervous system — were considerable hyperplasia of all the lymphatic glands, and some congestion at the bases of the papillae of the kidneys." Wilson succeeded also in recovering the organism from the cervical lymphatic glands in the monkey. However, Key and Retzius have shown that pigment injected into the subarachnoid space is to be found later in the cervical glands; hence, one must consider the possibility that these organisms may just as well have gone from the central nervous axis to the cervical glands as the other way. Certainly this is the direction of drainage.

However, when the organisms reach the central nervous system they produce there two definite changes: 1. In the mesodermal elements of the arachnoid and pia the result is edema and hemorrhage, with some increase of connective tissue cells, without any of the usual signs of inflammation, i. e., infiltration with white blood cells and lymphatic elements. The lymph spaces of the spinal blood vessels show no changes. 2. The changes in the ectodermal elements appear to be most marked in the neighborhood where the greatest mesodermal change takes place, i. e., in the arachnoid where the nerve roots run through it. These nerve fibers here show marked degeneration of a toxic and infectious nature. In the peripheral nerves the degeneration may be looked on as both secondary and toxic in nature. In the spinal cord and posterior root ganglions we see the usual picture of infectious-toxic degeneration, with some evidence of early secondary degeneration due to the destruction of the fiber in the arachnoid. The increase of the neuroglia cells, the degeneration of the nerve cells and the neuronophagy are all parts of the infectious-toxic picture. That the changes observed in Case 2 were, on the whole, more severe than in Case 1 is explained by the fact that the disease had had a longer course. In Bashford's cases where the changes were more severe than mine, this is probably due to the same cause.

The question of the proper name for the disease is still an open one. Gordon Holmes called it "acute infectious polyneuritis." Bradford, Bashford and Wilson have employed the same term with considerable

misgivings in the light of the central nervous system changes. Kennedy, recognizing the inaccuracy of the term, has suggested the name "acute neuronitis," not without misgivings on his part. The present writer is at a loss to name the disease properly. To call it an acute arachnoiditis appears very inadequate, and hence he has been obliged to fall back on the clinical manifestations of the disease for the title of this paper. Perhaps the term "acute infective meningomyeloneuritis might be acceptable.

INFECTIVE NEURONITIS*

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During the spring and summer of 1917, and to a lesser degree in 1918, I was privileged by circumstances in France to see a considerable number of cases having many of the easily recognizable symptoms of acute polyneuritis, that presented unmistakable evidences of involvement of the spinal roots and of the central nervous system as well.

Detailed reports of some of these, together with the necropsy material of two fatal cases that had been clinically carefully examined, I gave to Sir John Rose Bradford who collaborated with Captain Bashford and Captain Wilson in examining them along with other material arriving subsequently from other areas.

The brilliant results of these joint investigations have recently arrived in this country[1] and make it unnecessary to put before you an extended succession of clinical descriptions of this disease.

Sufficient stress, however, has not been laid on the clinical manifestations in these cases of the widespread affection of nervous tissue other than the lower motor neuron.

I propose, therefore, to describe certain cases under my care revealing definite clinical variants from the syndrome of polyneuritis as we have known it, which, when considered along with the widespread changes in the posterior ganglions, spinal roots, ventral cornual cells and Betz cells of the cortex, would seem to make it fitting to remove this disease from the neuritides proper, and to designate it by a title more descriptive of the clinical and pathologic picture produced.

REPORT OF CASES

CASE 1.—Private, J. J. W., Twenty-Fifth Northumberland Fusileers, when in good health suffered prolonged burial on May 28, 1917. He was shaken by this experience; when being sent down the line he had severe headache, and was tremulous and nervous; he complained of pains in his back and limbs and for four days had a mild rise of temperature. He then recovered from these generic symptoms, was made a temporary orderly in hospital, and did his work well and enjoyed it.

On July 14 he felt well and worked as usual. When handing out dinners in a ward he suddenly felt weak "in the thighs, not in the feet." He lay

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*Read before the Meeting of the American Neurological Association, at Atlantic City, N. J., June 16-18, 1919.

1. Quart. J. Med. **12**:88, 104 (Oct.) 1918 (Jan.) 1919.

down for two hours, after which he found himself hardly able to stand and experienced numbness and tingling in the toes; he found that he "could not feel his blanket with his toe ends."

At this time also he became conscious of similar sensations in the fingers. In a few hours he developed acute headache and shooting pain in the back of the neck, and aching pains in both knees. During these hours the feeling of numbness gradually crept up to the knees.

Next morning he had great difficulty in standing and found his finger tips anesthetic when washing. On that day the knee jerks were very brisk and the ankle jerks were absent. There was great weakness of the movements of both thighs and to a less relative degree of dorsiflexion of the feet. The arms, especially in shoulder movements, were weak; the face and palate were normal. He could, with difficulty, walk alone. Pinprick and touch could not be felt below the root of the toes on both feet and the middle of the first phalanx in the hands.

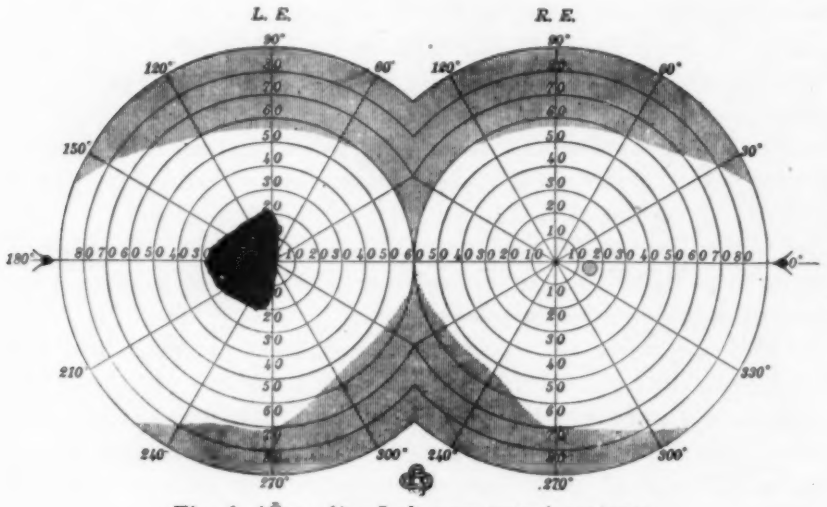

Fig. 1 (Case 1).—Left paracentral scotoma.

On the following morning the knee jerks were absent, the palate and diaphragm normal, and there was visible a very slight weakness in closing the left eyelid and in retracting the left angle of the mouth. The upper limit of analgesia was now across the dorsa of the feet and the middle of the hands, and there was progressive loss of strength in the hip and shoulder movements, without any corresponding change in distal power. On the afternoon of the same day he could not close the left eye, and he complained of inability to taste his food and of numbness in the mouth and lips. He was seen to have lost appreciation of pinprick and touch in both fifth nerve areas. The calf muscles were then very sensitive to pressure.

The next day (July 17) brought to him great difficulty in swallowing solids but not fluids—which difficulty was referred by him to below the level of the thyroid cartilage. (All these patients volunteered a description of this symptom, complaining of "a lump" being felt in the upper esophagus.) Dorsiflexion of the feet was still good, but gluteal, psoas and adductor actions were practically abolished. The right thigh was less affected than the left. The

muscles lacked tone, but were not wasted. He could feed himself easily because the hands were still strong, but movements of biceps, triceps, deltoid and trapezius were almost quite ineffective on both sides.

The zone of sensory loss extended below the upper third on the legs and forearms. He complained of difficulty in passing water, of not feeling the act of micturition and of not feeling the bedpan properly. Examination thereupon revealed an almost complete loss of sensation over a saddle-shaped area on the buttocks, and over the penis and scrotum as well. The diaphragm was hardly in action; the abdominal rectal muscles were very weak; he was quite unable to turn over in bed, and no abdominal reflexes could be obtained.

Five hours after this examination—on July 17 at 11 p. m.—the border of loss to pinprick and touch was in the middle of the thighs and the lower third of the upper arms. Deep muscle pain sense in the calves, sense of position in the toes, ankles, fingers and wrists, together with vibration sense in both tibiae, were all absent. The palate remained unaffected, while swallowing became progressively more difficult. Coughing became troublesome; there was also progressive weakness in getting rid of phlegm properly. He was unable to move at the hips or knees; the ankle and toe movements were present though feeble.

On the following morning, seven hours later, he was astonishingly better. He swallowed more easily. The diaphragm acted well. He could feed himself again, and could draw the legs up with fair power. Urine was passed with less delay than before, and he was dimly aware of its passage through the urethra. The sensory loss in the sacral root areas was still present but was less deep. The fifth nerve change was stationary, but the stocking loss of sensation was quite definitely reduced to below a sharply demarcated line two inches above the malleoli. On this day of improved condition the visual fields were mapped out, and a paracentral scotoma was found on the left side, accompanied by disk pallor and subjective loss of visual acuity in the left eye. This extraordinary rally was maintained for just ten hours when he suddenly began to feel weak, and in fifteen minutes lost all power of movement in the legs; inside of an hour he lost the power of phonation, and began to have intense difficulty in swallowing. There was great discomfort referred to below thyroid cartilage. The pulse rate was 134. For the first time there was weakness of the right side of the face. His voice could only be raised to a faint whisper, but he asked for a pencil, which was used clumsily but legibly. The diaphragm was not paralyzed; the palatal reflex was brisk; recti abdominis, fair. He could move his toes and ankles fairly well, but there was no visible movement achieved at the great joints. There was urinary retention and sacral sensory loss as before, but only the toes were then anesthetic, and there was no loss to pinpricks on the hands.

On July 20—the next day—the right side of the face was entirely paralyzed; otherwise his condition was somewhat better. He could occasionally phonate. The vocal cords were in cadaveric position and almost quite fixed. The corneae were anesthetic, the pulse slower and intermittent. Consciousness was acutely clear.

On July 21 he became cyanosed and died of asphyxiation. Blood cultures—by the ordinary methods—were in this and two other cases negative.

Examination of the cerebrospinal fluid was negative on the third day of his acute illness, at which time he had a leukocytosis of 14,000. Sections of the cortex, medulla and cord, cervical and lumbar enlargements with root ganglions and certain peripheral nerves were sent to Captain Bashford, to whose findings reference will be made later.

As outstanding features of this case one must notice: first, generalized peripheral neuritis; second, spinal root involvement as shown by diminished sphincter control and loss of sensation in the areas of distribution of the lower sacral segments; third, the changes in the posterior tracts which abolished appreciation of pressure on muscles, previously very sensitve in this regard, and which caused the senses of vibration and joint position to disappear.

The greater incidence of the disease on the proximal rather than the distal muscles is quite unlike the usual clinical picture of polyneuritis and is, I believe, due to the imposition of a palsy of ventral root and ventral horn origin on one proceeding from a patchy degeneration of peripheral nerves.

The constant paralysis of the face, and the constant sensation of obstruction in the throat may be regarded as due to specific action of the infecting agent and so are analogous to trismus in tetanic, and oculomotor palsy in diphtheritic infections.

Case 2.—Private H., Fifteenth Canadians, gave a history of having had "P. U. O." or "Trench Fever" some weeks before the onset of numbness and tingling in the hands and feet. When seen on Dec. 16, 1917, the right side of his face was paralyzed, his voice was very weak and the vocal cords were almost immobile in the cadaveric position. He experienced great difficulty in swallowing although the palate and its reflex were normal. There were extensive flaccid palsies around the shoulder joints, more grossly evident on the right than on the left side, and there was weakness but no paralysis of the hands. He was unable to turn in bed. The upper recti abdominis were definitely stronger than the lower recti. There was complete flaccid palsy at both hip joints, and almost complete palsy of both feet. He was quite unable to tell when micturition had begun or had ended. There was frequent bladder incontinence. All other spinal reflexes were lost. There was total loss of sensation in the fifth, sixth and seventh cervical root skin areas, and much relative loss in all the sacral root skin areas. My notes contain no mention, nor does my memory tell me of sensory defects referable to peripheral nerve change such as were described in the first case.

For the privilege of having seen this man and also the following I am indebted to the courtesy of Major Clayton, R. A. M. C.

Case 3.—Second Lieutenant C. E. C., Thirty-Third Battalion, Australian Imperial Force, was utterly exhausted at Paschendaele in October, 1917; he was ill and miserable, but did not go off duty. A month later he had what was thought to be influenza; three days afterward weakness in the legs developed, and within a few hours he was quite unable to stand. When I examined him a month later I found complete facial diplegia, difficult deglutition and as usual, a normal palate. There was flaccid palsy round the great joints of all four limbs with a high degree of atrophy in the gluteal and thigh muscles; all movements of the toes and ankles were retained and were of fair power. The trunk muscles were very weak, but the recti abdominis were strong and the abdominal reflexes were retained while all others were absent.

Pinprick and touch were not appreciated in the second and third lumbar root areas, but were acutely felt in the legs and over the sacral region. In over a month's illness he had only once lost control of his bladder.

Case 4.—The last case which it seems necessary to describe is that of Corporal H. H. E., Royal Army Service Corps, Divisional Train, who gave the usual history of a febrile attack in March, 1918, through which he had

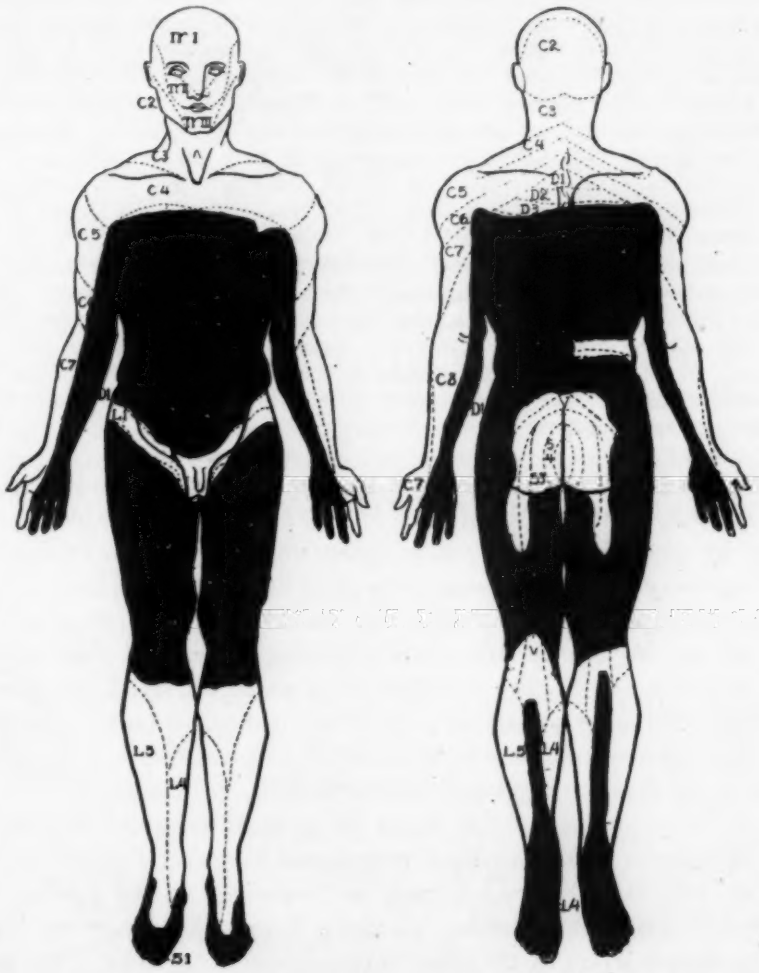

Fig. 2 (Case 4).—Shaded areas represent sensory loss.

remained on duty. Five weeks later numbness and tingling in the feet and legs and hands supervened. These were slowly progressive so that when I first examined him he was quite unable to walk. At the same time he showed an incomplete paralysis of the left face, and complained frequently of a feeling as though he had a lump in the throat although swallowing and speech were both functionally intact. The shoulder muscles were reduced in power, the grasps very weak. The trunk muscles were much enfeebled so that he could

not turn in bed without help. The thigh muscles were weak and flaccid and the glutei weaker and still more flaccid. The muscles below the knee were reduced in power to a much smaller extent.

The abdominal reflexes were lost. The knee jerks faintly present, the ankle jerks brisk and the plantar reflexes were both of extensor type. The sphincters were never affected, but no erections occurred, and all sexual desire was absent during his illness.

The sensory changes were remarkable and varied little in amplitude and not at all in distribution during the two and a half months he remained in hospital in France. There was almost complete insensitiveness to pinprick, touch and temperatures in the seventh and eighth cervical root areas, in all those served by the dorsal roots, and those supplied by the second and third lumbar and the first sacral roots; that is to say, from the seventh cervical segment downward, all the posterior root areas were almost quite defunctionated with the exception of the first lumbar, the fourth and fifth lumbar, and the second, third, fourth and fifth sacral areas, which were intact so far as repeated examinations revealed.

There was no loss of sense of position; deep muscle pain sense was certainly nowhere increased and was probably below normal in affected regions. The vibrations of a tuning-fork were not felt in the toes but were felt faintly on the patellae. They were, however, clearly felt on the tibiae and iliac crests. In affected areas, appreciation of compasses was absolutely bad, and elsewhere normal. The cerebro-spinal fluid was unchanged and the Wassermann test was negative.

While in my earlier cases the main incidences of the disease fell peripherally, with occasional signs of root involvement, produced probably by ascending lymphogenous extension, the last case emphasized the necessity of including something more than mere peripheral change in our conception of the condition as a morbid entity. Though its syndrome was necessarily determined somewhat differently from that of the others, yet its identity of origin, as shown by the facial, pharyngeal, humeral and other palsies, is indubitable; it displays nudely a clinical picture which was in the others, but which was overlaid as in a palimpsest by the detail of a generalized polyneuritis.

A study of the sections made by Captain Bashford revealed a patchy neuritis in the peripheral nerves, and a degeneration of the cells in the ventral and dorsal cornua, and especially in the cells of the posterior ganglions. Similar, but more benign changes, were found in the deeper layers of the cerebral cortex and in the cells of the pontine nuclei. A small round-celled infiltration was found around the ganglion and cornual cells, but never around the meningeal vessels and capillaries as is usual in poliomyelitis; this was, in Bashford's opinion, a much later phenomenon than were the degenerative changes in the nerve cells themselves.

The ependyma of the spinal central canal showed constant extensive proliferation. The meninges were normal.

Small quantities of an emulsion of affected spinal cord preserved in glycerin injected into monkeys subdurally produced the disease clinically after an incubation period of from five to seven weeks, and histologic examination of the nervous tissues of these animals revealed conditions essentially identical with those described in man. The disease further was reproduced by inoculation from monkey to monkey.

Nervous tissue from fatal cases and also from monkeys affected with the disease as has just been described were investigated bacteriologically by Capt. J. A. Wilson, R. A. M. C., who succeeded in obtaining positive cultural results by following the methods adopted by Flexner and Noguchi in their search for the organism of poliomyelitis. It suffices to state here that, under strict anaerobic conditions, yellowish colonies appeared capable of subculture composed of minute rounded bodies arranged irregularly or in pairs.

This organism inoculated subdurally into a monkey reproduced the disease clinically and pathologically, and was recovered later from the nervous tissues of the animal so inoculated.

In conclusion, stress must be laid on Wilson's observation of the ease with which the organism was recovered from the cerebral cortex — a consideration that lends emphasis to the present contention that here we have to do with a widespread neuronic infection and not with a condition limited to peripheral twigs.

HEREDITARY OCCURRENCE OF HYPOTHYROIDISM WITH DYSTROPHIES OF THE NAILS AND HAIR*

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The study of individuals and families that may show various manifestations of mental and nervous degeneracy not infrequently discloses the existence of constitutional disorders that are direct or indirect causes of these.

These constitutional disorders appear sometimes as peculiar defects or abnormalities of body structures that tend to be transmitted through the family. Numerous instances of these are recorded in the literature dealing with problems of heredity. These are in most instances treated rather more from the point of view of being specific unit qualities that are transmitted than as the results of a more general inherent deficiency of organs whose healthy functioning is essential for determining normal development and successful adaptation to the requirements of life.

Recently we have had the opportunity of studying a family that for generations had shown among its members peculiar trophic disorders of the nails and hair, and various abnormalities of a mental or nervous character.

While the more obvious abnormalities of this group are of special dermatologic interest, the fact that these seem to be fundamentally related to some constitutional disorder of an endocrinopathic type that peculiarly affects the nervous and mental make-up of the members of this family, makes the entire subject one of much importance for neuropsychiatry.

Our acquaintance with this family began with a boy who, at the age of 18, had been brought into the Juvenile Court of Detroit because of several instances of petty thieving. The frequency of his delinquencies and certain mental traits suggested the existence of feeblemindedness and led to his admission for observation to the Psychopathic Hospital at Ann Arbor.

REPORT OF A CASE

Examination.—The physical appearance of the boy was quite abnormal (Figs. 1 and 2). He was about 5 feet, 5 inches tall, and weighed 138 pounds. He appeared unusually well nourished. The skin of the face, trunk and

* Read before the meeting of the American Neurological Association held at Atlantic City, N. J., June 17-18, 1919.

extremities was plump and felt as if it were thickened in its deeper parts. Around the eyes the skin was puffy as if from increased subcutaneous fat. The hands were chubby and the fingers were short and clubbed. The hands and feet were cyanotic. The surface of the skin lacked moisture. The skin of the finger tips and soles of the feet was exfoliated in places.

A very striking feature in his appearance was the scant amount of hair on the head and body and peculiar abnormalities of the nails. While the head seemed fairly well covered by hair, this on close inspection was seen to be unusually thin, and there were patches in which the scalp was covered only by a fine lanugo. The eyebrows were reduced to a few scattered hairs and these were stiff and short. There was but little hair in the axillary and pubic regions and the latter conformed to the feminine type of arrangement.

The nails of the fingers and toes all lacked a quarter or a half inch of reaching the tips of the fingers or toes. There was no lunula. Their free margin was thickened and broken. Their surfaces were smooth and not ridged. The nail bed was exposed and in places showed suppuration (Fig. 3).

The pulse rate was 80; the blood pressure 130. There were no cardiac abnormalities. The body temperature was always a little below normal. On several occasions it was 96.8 F.; usually it was under 98. The Wassermann reaction test was negative on the blood. The urine at admission had a specific gravity of 1.010 and showed a few hyaline and granular casts. In other respects it was negative. The roentgen-ray examination showed a normal sella turcica.

There were a number of neurological abnormalities. There was a frequent tic-like movement of the face, and when he became emotionally excited there was a tic of the right shoulder. His speech was somewhat thick and nasal in quality. Bone conduction for sound was absent in the left ear. The left pupil was larger than the right. The fundi of the eyes were normal. Refraction tests showed a high degree of hyperopia. The form field of the left eye showed a retraction of the field for white. There were no paralyses or disturbances of sensibility. The fingers showed a fine tremor on extension.

His mental reactions were slow and he showed a definite intellectual defect. Psychometric tests gave him a mental age of 12 years and 6 months. His attitude toward his delinquencies was immature and unreasonable. In other respects he showed few definite peculiarities.

The general appearance of the boy, the peculiar condition of the skin and the dystrophies of the hair and nails suggested the existence of a myxedematous condition. This view was confirmed by a biopsy of the skin kindly made by Professor Wile of the department of dermatology.

The patient was tested for glucose tolerance and it was found that he could take 250 gm. of glucose in twenty-four hours before sugar appeared in the urine.

Treatment.—He was put on thyroid extract in 5 grain capsules three times a day. At the end of twenty-seven days there was increased perspiration. The exfoliation of the skin of the hands and feet ceased. The hair was less fragile and there was a very noticeable increase in the hair of the scalp and eyebrows. The nails appeared to be improved and they seemed to show some growth. Subjectively he appreciated an improvement in his general feelings. After a month of thyroid treatment he began to show toxic effects; the pulse rate was 110. Treatment was discontinued. The improvement of the skin condition at once ceased. The dryness and exfoliation again returned. On resuming treatment, improvement again occurred.

The clinical symptoms and their response to treatment in this case leave little doubt but that the disorder of the nails and hair were but parts of a constitutional disturbance due to hypothyroidism.

Family History.—The case became of increased interest through the fact that these same deficiences had been present among members of the boy's family for at least six generations, and that in addition to the dystrophies of the hair and nails, both those affected in this way and many others without these defects showed various disorders of nervous functions (Fig. 4).

It has been possible personally to substantiate the accuracy of the data in this case in almost every instance. Our information regarding the family goes back to the great-grandmother of our patient, I—1 M. J. C. 1st. This woman lived in Canada and was of French extraction. It is known definitely to members of the present generations that she had the same characteristic abnormalities of nails and hair. A photograph in the family shows a marked facial similarity to her daughter, II—1 M. J. C. 2nd. It is probable that these abnormalities were present in still earlier generations. This is suggested in the reply that M. J. C. 1st gave to her daughter that these were the signs of an unhealthy generation. Information that we regard as trustworthy, but that has not yet been personally confirmed, is that a brother of II-1 has the abnormality and of his six children, three have the disorder, and one is insane.

II-1 married a man who was free from defect. From this marriage there were ten children. The oldest of these, a male III-2 had the characteristic defect. He married a woman without the defect, and from this marriage there were six children. The oldest, a female, L, IV-1, has normal nails and fairly abundant hair of the scalp, but few in her eyebrows. She is of short stature and has peculiar short, chubby fingers. She is of neurotic constitution, and from early childhood has had a severe facial tic. She has had no children. The second child, a female, A, IV-2, does not have the defect. She is either hysterical or epileptic. When a small child she would have attacks in which she was unclear. In these she would take hold of her mother and remark that the floor was sinking under her. Later on these ceased, but at the age of 16 she had attacks in which she was unconscious for a brief period. There were no convulsive movements. These have recurred from time to time during her adult life. It is of interest to note that although she has always had an abundance of hair on her head, in the spring of 1919 she had a miscarriage, and following this her hair fell out in large amounts. She is married to a man without the defect and has had two children. The first of these died in infancy from pneumonia, and the second during the first years of her life suffered from marasmus. P, IV-4, a male, was our patient. His physical and mental disorders have been noted previously. It is said by his family that he and his father closely resembled each other, not alone in the defects of nails and hair but in having a thick skin and in their general physical make-up. Since his discharge from our observation, this boy has been delinquent frequently, having been implicated in several instances of stealing, and at the present time his whereabouts are unknown.

Between the third and fourth child there were two miscarriages. The fourth child, a boy, G., IV-5, is now aged 19. He has thin eyebrows and a fair amount of head hair. His nails are normal. During an attack of typhoid fever he had a severe delirium and since then he has never been physically strong.

The sixth child, a boy, E., IV-6, has the characteristic defect of nails and hair (Figs. 1 and 2). He was first seen at the age of 7. His appearance

Fig. 1.—Patients IV-4 and IV-6.

at that time had a cretenoid character. He looked like a little old man. His skin seemed myxedematous. There were patches of baldness on the top and back of the head and there were no eyebrows. Four years later his hair was uniformly scant over his head. There was a lateral scoliosis in the dorsal region. The thyroid gland was not palpable. There was a tic-like movement of the head and choreiform movements of the extremities. At the age of 13 he came into the courts because of malicious conduct and incorrigibility. He was sent to the State Training School for the Feebleminded. Psychometric examinations gave him a mental age of 9 years and 1 month. He responded very little to thyroid treatment.

The seventh child, R., IV-7, does not have the defect, but is mentally subnormal.

The second child of the third generation, W., III-3, a male, is said to have had hair at birth, but none after early childhood. At the present time, he is completely bald. He has no eyebrows or eyelashes and no body hair. His nails are defective in the characteristic way. Between the ages of 25 and 32 he had typical epileptic convulsions. At the present time he has peculiar feelings as if an attack were coming on, but no further development. He married a woman free from the defect and has had four children. The eldest, a girl, M., IV-8, has the defect. Her head hair is limited to a thin downy covering. Her nails are defective and she has thick skin and clubbed fingers. Mentally she is backward, and is now in a special room. Her thyroid is hard and abnormally small. Menstruation first appeared at the age of 16.

T., IV-9, a boy, aged 12, lacks the defect in the characteristic type, but his hair is unusually thin. He has nocturnal eneuresis. Mentally he is feebleminded.

H., IV-10, a boy, aged 10, does not have the defect, but is feebleminded.

J., IV-11, a boy, aged 5, has thin hair, but normal nails. He has nocturnal eneuresis.

The third child of the third generation, J., III-5, lacks the defect. He has had a bad criminal record in the courts. He has twice married, but has had no children.

The fourth child, E., III-7, seems to have been quite normal as to hair and nails. She married a normal man and all of her descendants, for two generations, have been free from the defect.

The fifth and sixth children of the third generation were twins and lacked the defect. One of these, III-9, died in infancy. The other, E., III-10, married a normal man and had two normal children. She, herself, died from acute tuberculosis.

The seventh child, L., III-2, had normal hair and nails. She married, and has had eleven children. All of these have remained free from the defect.

The eighth child, J., III-14, has no hair on her head and her nails are defective. She married a normal man, III-15, who is also a brother of III-4. She has had seven children. The first of these, T., IV-30, was free from the defect, but died in childhood from scarlet fever. The second and third children lacked the defect, but died in childhood from marasmus. The fourth child, W., IV-33, died in infancy from meningitis.

The fifth child, M., IV-34, a girl, aged 13, has normal nails and hair, but her teeth are notched along the cutting edge. Mentally she is feebleminded.

The sixth child, C., IV-35, has the typical defect. He has nocturnal eneuresis and is feebleminded.

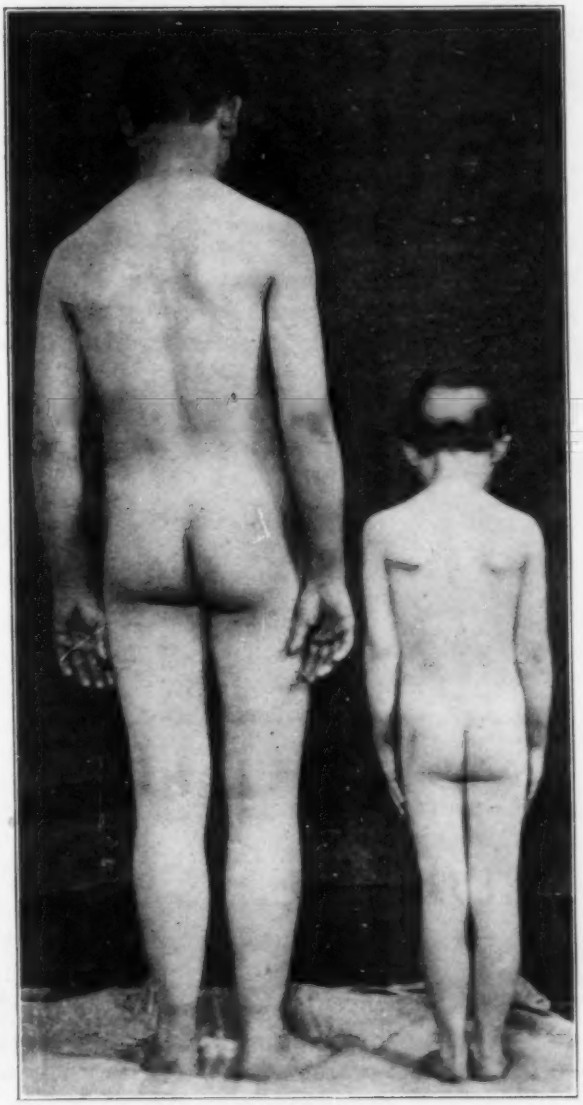

Fig. 2.—Patients IV-4 and IV-6.

The seventh child, M., IV-36, aged 7, has the family abnormality of nails and hair. She has nocturnal eneuresis and is feebleminded.

The ninth child of the third generation, M., III-16, lacks the defect. She married a normal man and has had nine children, all of whom are free from the defect and are of average mentality.

The tenth child, L., III-18, has the defect. Her teeth are worn away on the cutting edge. She married a normal man, and has had three children. The oldest of these, A., IV-46, has always had good hair, but his nails show the typical defect. He has some difficulty in hearing, but otherwise is healthy. The second child died in infancy and had abnormal nails. The third child, R., IV-48, has the defect. While the head seems fairly well covered by hair it is abnormally thin in amount. The teeth show irregularities of the cutting edge. Mentally he is subnormal.

Among sixty-one members of this family, belonging to six generations, the defect in its typical manifestations was present in fourteen instances. In these, both hair and nails were affected. The nail defect seemed to be about the same in all, but there was much variation in the

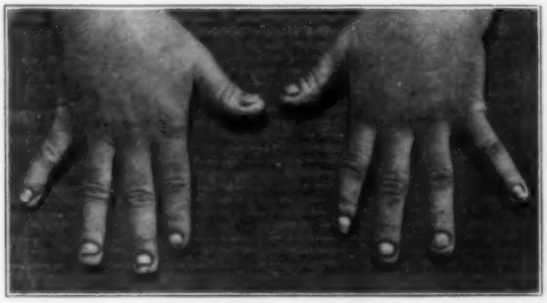

Fig. 3 (Patient IV-4).—Abnormalities of nails.

degrees of loss of head hair. In the greater number there was an extreme scantiness as to the amount of hair. In no instance was there a total loss of all hair of the head, the most extreme cases showing a fine lanugo-like covering of the scalp.

The defect tends to occur in a mendelian type of distribution, but the varied character of the abnormalities appearing in relation with the type defect of nails and hair is perhaps too complicated to be explained in a simple mendelian formula. The character of abnormal hair and defective nails behaves as a mendelian dominant. All persons that have the defect give a mixed progeny when crossed with normals. The defect never appears in a descendant who himself is free from the defect.

Aside from this particular abnormality, there are other features that show that in this family group there are conditions active in the production of a variety of disorders that are of much interest to neuropsychiatry. This is the high frequency of feeblemindedness and

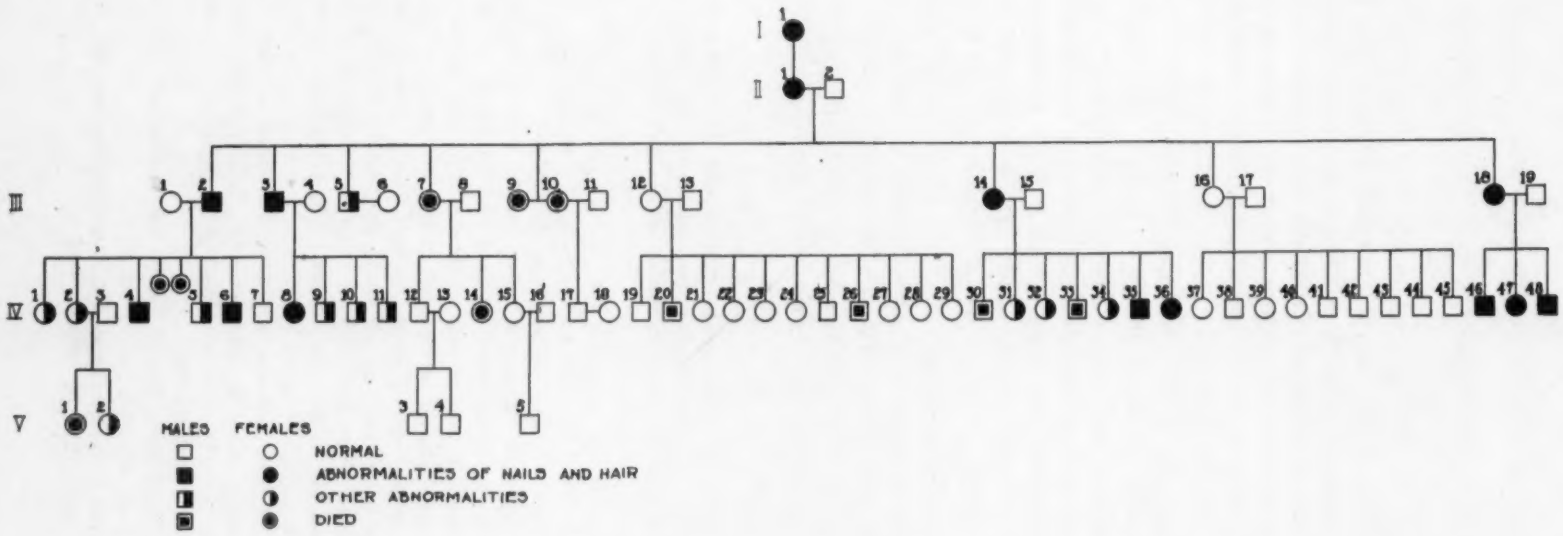

Fig. 4.

Heredity chart of family showing hypothyroidism and abnormalities of nails and hair.

neurological disorders of a degenerate type that are present among the family.

The members of the third generation who had dystrophies of nails and hair, and all of their descendants, numbered twenty-nine persons. Of these, twenty-two were definitely abnormal. Twelve of the latter had the characteristic family dystrophy, and ten others, who lacked this, showed other constitutional and nervous disorders. These included one case of epilepsy, one of hysteria, one of severe tic, four instances of feeblemindedness, one of nocturnal eneuresis and four died at an early age from marasmus. Even those who had the nail and hair dystrophy had other abnormalities. One of these was an epileptic; one had cancer; four were feebleminded, one had nocturnal eneuresis.

This multiplicity of characteristics of degeneracy bears out the view that we are concerned in this family with a more fundamental disorder than that of an isolated abnormality of nails and hair. It would seem that there was present some organic defect that had widely distributed constitutional influences.

The well-known association of abnormalities of hair and of nails in hypothyroidism and the two fairly well defined cases of juvenile myxedema, and the reaction of one case to thyroid feeding, seems to warrant the conclusion that the fundamental disorder in this family was of the thyroid gland.

Cases Reported in the Literature.—There are in the literature a few other observations on families showing similar dystrophies of nails and hair. The earliest one of these and the most extensive in its abnormalities was that reported by Nicolle and Hallipre.[1] In this family of fifty-five known persons, thirty-six showed the dystrophies. In 1896 White[2] reported the study of a family in which there were fourteen members in four generations. Of these, seven showed the characteristic abnormalities. Eisenstaedt,[3] in 1913, reported observations on a family of thirteen members which showed the occurrence of the dystrophies in five generations. Among eight members of the fourth generation, the abnormality was present in three. It is also probable that a contribution by Hoffman[4] should be included in this connection. This reports a line of thirteen persons, eight of whom through four generations had shown abnormally short and scant eyebrows and dystrophies of the nails of the hands and feet. In this family there were several instances of thyroid disease. The patient had a large struma, as had her mother. The appearance of the mother

1. Nicollé and Hallipré: Maladie familiale caracterisée par des alterations des cheveux et des ongles, Ann. de Derm. et Syph., 3d Ser. **6**:804, 1895.

2. White, Charles J.: Dystrophia Unguium et Pilorum, Hereditaria, J. Cut. and Gen. Urin. Dis. **14**:220, 1896.

3. Eisenstaedt, J. S.: Three Cases of Family Dystrophies of Hair and Nails, J. A. M. A. **60**:27 (Jan. 4) 1913.

4. Hoffman: Ueber Verkümmerung der Augenbrauen und der Nägel bei Thyroidosen, Arch. Derm. u. Syph. **89**:381, 1908.

suggested hypothyroidism and Hoffman comments that "perhaps one might believe that a dysthyroidism was the predisposing cause for the described malformations."

The frequency of disorders of the nails or of the hair in hypothyroidism is shown in several analyses of large groups of cases of myxedema. In an analysis of 150 cases, Hun and Prudden[5] found malformations of nails in 75 per cent. of cases studied, and in a later series Howard[6] found nails abnormal in 86 per cent. of cases, and hair in 93 per cent. of those studied.

As to the heredity of hypothyroid disorders, there is a good deal of confirmatory evidence. In the previously cited analyses of large groups of cases, Hun and Prudden found a direct inheritance in 8 per cent. of cases studied, and Howard in 6.6 per cent.

It would be of interest to know to what extent some of the so-called neurotic disorders of the hair are brought about through the reaction of the thyroid gland to disorder of the sympathetic nervous system coming primarily from affective disturbances such as the sudden loss of hair or change in color following fright and nervous stress. There may be persons whose thyroid functioning is just sufficient to maintain normal health, and under stress this balance may be so disturbed as to produce definite pathologic conditions. In this group we have reported there were two persons who lacked the characteristic family dystrophies, who, following slight constitutional disturbances suffered a loss of hair and later regained normal conditions.

State Psychopathic Hospital, University of Michigan.

5. Hun and Prudden: Myxedema, Am. J. M. Sc. **96**: July and August, 1888.
6. Howard, C. P.: Myxedema: A Study, J. A. M. A. **48**:1226 (April 13) 1907; ibid. **48**:1403 (April 27) 1907.

CONGENITAL FACIAL PARALYSIS*

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LITERATURE ON THE SUBJECT

The significance of cases of congenital facial paralysis is still a question of considerable interest. For the last fifty years clinical observations with scanty pathologic reports have been recorded.

Anatomists, even such as Piersol,[1] who discusses anomalies under "Practical Considerations," do not mention congenital facial paralysis. This is true to a less extent of pathologists. Some of them do not mention congenital facial paralysis (Adami and Nicholls,[2] Bruning and Schwalbe,[3] Monakow[4]). Others simply state that in gross maldevelopments of the brain the cranial nerves also suffer (Ernst[5]); while others admit and discuss the possibility of congenital facial paralysis with reservations; for instance, Ballantyne,[6] writing as follows: "Sometimes, as in a case about which I was consulted by Dr. Dickson of Lochgelly, 1899, the long persistence of the paralytic condition throws doubt upon the peripheral nature and traumatic origin of the palsy. Under these circumstances, it is reasonable to turn from an intranatal to an antenatal mode of origin of the nerve lesion. It may, then, be due to a lesion in the facial nuclei in the pons, or in the fibers connecting them with the cortical centers; but, of course, even

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* Read at the meeing of the American Neurological Association, Atlantic City, N. J., June 16, 1919.

1. Piersol, G. A.: Human Anatomy Including Structure and Development and Practical Considerations, Philadelphia, J. B. Lippincott Co., 1907.

2. Adami, J. G., and Nicholls, A. G.: The Principles of Pathology, **2**: Philadelphia, Lea & Febiger, 1909.

3. Bruning, H., and Schwalbe, E.: Handbuch der Allgemeinen Pathologie und der Pathologischen Anatomie des Kindesalter, **1**: 1912.

4. Monakow, C.: Gehirnpathologie, Ed. 2, 1905.

5. Ernst, P.: Missbildungen des Nervensystems, in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Part 3, No. 2, Section 2, Chapter II, pp. 100, 101, 251.

6. Ballantyne, J. W.: Antenatal Pathology and Hygiene, **1**: New York, William Wood & Co., 1902, p. 47.

this central origin, or cerebral form, may be of intranatal origin, although it is unlikely."

Congenital facial paralysis has escaped the notice of Gould and Pyle,[7] who discuss many rare and curious anomalies. It is not mentioned in Nouvelle Iconographie de la Salpetriere, 1877 to 1915.

Clinically, on the other hand, the existence of congenital facial paralysis has been definitely established, but many authors of systematic works do not mention it (Taylor,[8] Burns, Cramer and Ziehen,[9] Thompson,[10] Turner[11]). Others simply state the fact that congenital defect of the temporal bone causes facial paralysis (Jelliffe and Whitehall[12]), and only a few clinicians discuss cases reported in literature, for instance, Oppenheim,[13] who states that the condition is associated with ocular palsies, deafness, defects of muscles and temporal bone, etc. The same is true of Vogt,[14] whose remarks, however, are interesting, viz.; "In peripheral facial paralysis the whole side of the face is motionless; in central and in most of the bulbar (types) the superior branch retains its movement. In congenital cases the superior branch remains motionless, while the region about the mouth retains either complete or partial motility. When this difference between the upper and lower part of the face is great, it gives a characteristic facial expression: full, protruding lips which are in contrast with the mask-like, expressionless, sunken face above. This appearance of the face makes the condition often recognizable at the time of birth, and gives one the impression of an advanced atrophy or aplasia of muscles; also the skin over the muscles is usually smooth, peculiarly shiney, and colorless."

In periodical literature quite a number of cases have been reported. As early as 1898 an excellent paper was read before the American

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7. Gould, G. M., and Pyle, W. L.: Anomalies and Congenital Curiosities of Medicine, Philadelphia, W. B. Saunders Co., 1897.

8. Taylor, E. W.: Case Histories in Neurology, 1912, in Diseases of the Cerebral Nerves. Osler's System of Medicine, Mod. Med. **5**:471, 1915.

9. Bruns, L., Cramer, A., and Ziehen, T.: Handbuch der Nervenkrankheiten in Kindesalter, Berlin, 1912.

10. Thomson, H. C.: Diseases of the Nervous System, Ed. 2, New York, Funk & Wagnalls Co., 1915.

11. Turner, W. A.: Diseases of the Cranial Nerves, in System of Medicine, Allbutt & Rolleston, London **7**:500, 1910.

12. Jelliffe, S. E., and White, W. A.: Nervous and Mental Diseases, Ed. 2, Philadelphia, Lea & Febiger, 1917, p. 291.

13. Oppenheim, H.: Lehrbuch die Nervenkrankheiten, Ed. 6, Berlin, S. Karger, 1913, p. 630.

14. Vogt, H.: Infantile Beweglichkeitsdefekte im Bereich der Hirnnerven, Handbuch der Neurologie (M. Lewandowsky), Berlin, 1911, p. 270.

Neurological Association by Dr. H. C. Thomas,[15] based on the study of two brothers. Dr. Thomas reviewed the literature in detail and, concerning his cases, summarized as follows: "That the muscle defect in these cases is a congenital one, I think cannot be doubted. In the first place, the mother is quite sure that the defect was present when the children were born, although she cannot state that she noticed the deformity immediately after birth. The fact that the malformations occurred in two members of the same family, and that it is bilateral, speaks for its congenital origin, and the occurrence of other faults in development, the misshaped ears and possibly the deafness, lends added weight to this view. The character of the paralysis itself is quite similar to that which is found in the other congenital cases."

In recent years communications on this subject have appeared from Langdon,[16] Hutchinson,[17] Rainy and Fowler,[18] Batten,[19] Harris,[20] Neurath,[21] and others. Heller,[22] summarized in his Paris thesis the relations of congenital facial paralysis to agenesis of the temporal bone. His conclusions are as follows: "There is a special variety of congenital facial paralysis, which is caused by agenesis of the temporal bone. It is due to arrested development of the petrous portion and portions of the auditory apparatus.

"It may be complete, with total loss of voluntary muscular contractions and of electrical reaction, or partial, with preservation of the function and characteristics of the facial nerve."

To this type belong the cases of Harris[20] and Neuenborn.[23]

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15. Thomas, H. M.: Congenital Facial Paralysis, J. Nerv. & Ment. Dis. **25**:571, 1898.

16. Langdon, F. M.: Facial Paralysis, Congenital, Unilateral and of Unique Distribution, J. Nerv. & Ment. Dis. **26**:593, 1899.

17. Hutchinson, Sir J.: Persisting Paralysis of the Muscles Supplied by the Facial Nerve, Arch. Surg. **2**:20, 1900.

18. Rainy, H., and Fowler, J. F.: Congenital Facial Diplegia Due to Nuclear Lesion, Rev. Neurol. & Psychiat., 1903, p. 149.

19. Batten, F. E.: Congenital Affection of Sixth, Seventh and Twelfth Cranial Nuclei, Brain **28**:359, 1905.

20. Harris, H. E.: Congenital Absence of the Left Ear with Facial Paralysis, Brit. J. Child. Dis. **1**:502, 1904.

21. Neurath, R.: Zur Frage der Angeborenen Funktionsdefekte in Gebiete der Hirnnerven, München. med. Wchnschr. **54**:1224, 1907.

22. Heller, A.: Contribution a l'Etude des Paralysies Faciales Congenitales, par Agenesie du Rocher. Theses de Paris, 1903.

23. Neuenborn, R.: Rudimentaer Entwickelte Missbildete Ohrmuschel mit Congenitaler Einseitiger Facialisahmung Infolge Hypolasie des Nerven, Arch. f. Ohren **63**:58, 1904.

Recently the maldevelopments of the ear have been studied by Marx,[24] who wrote a profusely illustrated chapter in Schwalbe's Morphologie der Missbildungen, in which he touches on congenital facial paralysis. He believes this condition is mostly secondary to anatomic defects of the middle ear, but in some instances due to primary hypoplasia or agenesis of the facial nerve. This, according to Neurath,[21] practically summarizes all that is known about the pathology of congenital facial paralysis, and we may add that it is frequently associated with many other congenital anomalies. To correlate these anomalies pathogenically is often very difficult. He says: "The existence of congenital nuclear aplasia, although rare, has been definitely established" (Zappert[25]). Supporting this statement is the instance by Rainy and Fowler,[18] who found that "the facial nerve on both sides showed very marked degeneration, alike in the ascending part of the root, in the fasciculus teres, and in the emergent portions. Degenerated fibers could also be clearly traced passing through the healthy sixth muscle in the manner described many years ago by Gowers and von Gudden." The facial nuclei were also found to be pathologic.

"In a number of cases, especially in paralysis in the region of the eye muscles and of the facial nerve, a peripheral affection (muscle, nerve) is proven" (Zappert[25]).

REPORT OF CASE

Physical Examination.—W. B., female, aged 11, was sent to Dr. Fry for examination by Dr. R. N. Crews, Fulton, Mo., June 27, 1918.

The facial palsy of both sides is evident at a glance. Only a few minutes' inspection is necessary to reveal that it is complete and total. There is no motility whatever, except a slight drawing down at the corners of the mouth; this is evidently accomplished by the platysmata and is a little more pronounced on the left side. She is a well nourished, healthy child, with good complexion and hair, yet the absence of animation in her countenance is striking. When instructed to close her eyes, the mask effect is complete, the clear sclera presenting through a palpebral fissure a little over a quarter inch wide on each side. The skin is clear and smooth over the whole face, and only slightly thinned over the forehead and over the malars and nose. There is no corrugation, with the exception of a slight corrugation about the eyes and mouth. The upper lip is thin and slightly pursed, and the lower slightly everted. There is very little drooling and only when speaking or eating.

There are no lateral movements of either eyeball. There is a good range of vertical movement, and it is the same for each eye. The downward range is greater and seems quite full; the upper range is possibly somewhat limited.

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24. Marx, H.: Die Missbildungen des Ohres, in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, **3**: Part 3, No. 2, Section 2, p. 595.

25. Zappert, J.: Ueber Infantilen Kernschwund Ergebnisse der Innerer Medicine und Kinderheilkunde **5**:305, 1910.

There are no oblique movements, or, if any, so slight as to be questionable. The pupils are equal, and react well to light and to accommodation. The ciliary function is intact. In making the above observation, we had the valuable assistance of Dr. Joseph W. Charles, who joined us in examining the eyes.

There is no lateral movement of the mandible. The members of her family seem quite aware of this, and her father, who accompanied her, offered to

Fig. 1.—A girl, aged 11, in whom there is a complete absence of mobility in the distribution of the seventh nerve on both sides of the face. There is also an absence of all lateral movements of the eyeballs, mandible and tongue, and a teratologic absence of the left breast.

demonstrate her manner of masticating by furnishing her some gum to chew. The range of vertical movement of the jaw is possibly somewhat limited, but not much, and the strength of contraction seemed full enough.

The tongue is atrophied and corrugated; somewhat more so on the left. Its movements are limited and wobbly.

The range of movement in the soft palate and uvula is quite limited. It also has a corrugated appearance, more so on the left. The whole faucial surface seemed to be exceedingly sensitive, making the examination of it difficult, although the patient tried to acquiesce. There seems, however, to be little, if any, difficulty in deglutition. There are no motor defects in other portions of her body. The reflexes generally present no abnormalities.

Sensibility everywhere seems intact and well up to normal standard, including the special senses and general sensibility.

Mental Examination.—In intelligence and character this little girl seems quite up to the average of her age. Her speech is, of course, very defective; in fact even to her father it is almost unintelligible. However, her brother and sister and her teacher understand her "lingo" very well. She does not use standard sign language, and, although she writes very well indeed for one of her age, she does not converse in this way to any extent; nor does she use much pantomime beyond some coy little movement of her shoulders and hands for expressional purposes.

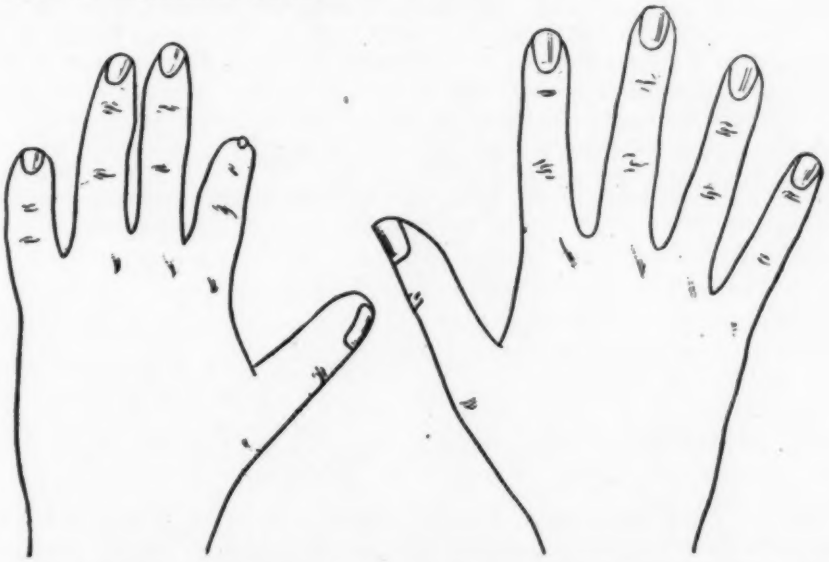

Fig. 2.—This figure shows the relative size of the right and left hands and the teratologic deformity of the left.

Although her face is so utterly expressionless (the more so on account of the limited eye movements), and her speech very defective, her demeanor is otherwise so alert that one is quickly assured of her intelligence. In the record are specimens of her handwriting and figuring that are very creditable.

History.—The parents have always been aware that the defects in the child were congenital; they have also believed they were hereditary, for there is in the family a tradition at least, that all the members of the mother's family have a webbing of the second and third toes, and a report that the mother's father had a withered arm from birth and that two of the mother's brothers had congenital deformities. The details, however, were not obtainable.

The mother and father of our subject and their other children present no abnormal features. They are all "healthy and well."

At the birth of W. B. (our subject) the delivery was precipitous, and the mother was unattended by a physician for an hour after the child was born, but there is no record of any untoward result to the child. When she was a year old her health seemed perfect. In her second year she had pneumonia, and for several years thereafter was rather delicate, especially suffering with conjunctivitis, which resisted ordinary methods of treatment. By the time she was 8, however, she was well and strong. In her ninth year her tonsils and adenoids were removed. Since she has been perfectly well, "full of life, jolly, and an excellent student."

The left mamma is entirely absent, the merest semblance of a nipple persisting. She seems to have fair strength in the pectoral muscles of this side, but the flat, attenuated appearance of the breast in contrast with the other side, gives the right side the appearance of being over-developed for her age.

The appearance of the left hand is so evidently teratologic, that only a brief description is necessary. The index and little finger are so short as to be quite out of proportion to the other digits. The whole hand is considerably smaller than the right. There is no atrophy, and she uses it very well, but not so freely as the right. The muscles of the arm are all present and proportionate, although the member is somewhat smaller than the right, both in length and general circumference.

We shall not in this connection attempt to discuss the teratologic bearings of the case, more than to suggest that the deficiencies here present are of the type due to germinal defect as distinct from the teratologic types caused by mechanical process in utero, as, for example, club-foot, cyclopia, spina bifida, etc.

PROBLEMS IN THE DIAGNOSIS AND TREATMENT OF INJURIES TO THE PERIPHERAL NERVES

THE OUTLOOK FOR THE FUTURE *

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This paper, based on observations made on 170 patients with injuries of the peripheral nerves whom we have studied at U. S. Army General Hospital No. 1, deals only with some problems which we have encountered and with the means and methods by which we have attempted to solve them. The paper concerns itself somewhat with the diagnosis of the injuries, but chiefly with the general conditions on which successful treatment must depend. We have not allowed ourselves to draw any conclusions as to the final results of the methods of treatment used as several years must elapse before conclusive data can be forthcoming. We shall, in what follows, speak of the significance of some symptoms and signs of nerve injury, of some aspects of the finer anatomic structure of the peripheral nerves, and of the anatomic basis and guiding principles for the technic of nerve suture and nerve grafting.

I. THE SIGNIFICANCE OF SOME NEUROLOGICAL SYMPTOMS AND SIGNS OBSERVED IN NERVE INJURIES

1. *The "Tinel" Sign.*—Much has been written concerning the importance of the tingling that can be observed when the skin is tapped along the course of a peripheral nerve. According to Tinel, tapping distally to the site of the nerve injury causes tingling referred to the area of distribution of the nerve only when the nerve in question is regenerating. On the other hand, tingling cannot be observed when a peripheral (mixed) nerve has been completely divided. Tinel claims also that in a regenerating nerve the tingling can be obtained on tapping at the points along the nerve more and more toward the periphery corresponding to the gradual downgrowth of the axis cylinders.

We are not yet in a position to deny or affirm that formication obtained by tapping on the nerve distally to the point of injury occurs

* Read at the meeting of the American Neurological Association, Atlantic City, N. J., June 16, 1919.

only, or invariably, when sensory fibers are regenerating. The value of the sign from the practical diagnostic standpoint is doubtful. We have in not a few cases observed this distal formication in patients in whom operation later showed a defect in the nerve with no discoverable (macroscopic) fibers bridging the gap. But even though the sign definitely indicated regeneration of sensory fibers, this does not necessarily mean that regeneration of motor fibers has occurred or will occur. Practically the necessity for surgical treatment was decided in every one of our patients by the behavior of the motor fibers. If motor fibers were not regenerating in sufficient numbers by the sixth to the eighth months after the injury, whether the sensory fibers were functioning well or not, it was assumed that a serious impediment was obstructing their progress, and exploration was advised. In all except two of the cases of this series (to be mentioned below) in which operation was advised and carried out, either complete severance of the nerve was found, or such involvement in scar tissue or fraying of the nerve, as made spontaneous progress impossible.

2. *The Skin Area Over Which a Given Sensory Nerve is Distributed Varies Considerably in Size in Different Individuals.*—Thus, if sensation remains normal in the superficial radial area in patients with musculospiral injuries, one must not be oversanguine in hoping therefore to find an incomplete severance of the nerve. In some persons the area supplied by the superficial radial is, like that of the anterior tibial nerve in the foot, only a penny-sized spot. Similarly, in one of our patients with a sciatic injury, there was merely reduction of sensibility in the musculocutaneous area of the external popliteal nerve, and we had expected accordingly to find some sound fibers passing through the damaged portion of the sciatic nerve. But the operation showed the upper stump of the external popliteal bundle of the sciatic disconnected from the distal stump and driven back nearly at a right angle to its normal position. The area was presumably supplied by overlapping fibers from neighboring nerves.

In making inferences based on sensory findings one must, therefore, keep in mind these individual variations as to distribution, and that the overlapping fibers from contiguous nerves gain in acuity of perception as the patient is forced to depend on them. Whether or not the overlapping fibers may actually progress farther into a defective area as time elapses after the injury, we are unable to say from any experience gained in the present study.

3. *Symptoms and Changes in Nerves Due to Ischemia.*—In four of our patients, in addition to the injury to the nerve, the main blood vessels of the limb had been severed when the wound was inflicted, or closed by the subsequent contraction of scar tissue. In many the

nutrient arteries to the nerves had been destroyed. And in most patients whose wounds were in nerves that lay adjacent to large blood vessels, the blood supply of the limb distal to the wound was reduced by pressure on the vessels.

Muscles and nerves respond promptly to quantitative or qualitative changes in the blood because of the complexity of the biochemical changes that occur in them. In the case of nerve fibers, their attenuated form and their dependence on the distant cell bodies for trophic control cause them to suffer doubly under the conditions mentioned above. These patients showed symptoms that were not confined to the distribution of the nerves that had been wounded. A general reduction of sensation was found throughout the limb. The muscles controlled by other nerves were weak and atrophic. Coldness of the skin, subjective and objective, and cyanosis in some cases, were present.

Because of the frequency of this factor of ischemia from pressure on blood vessels, we became more confident as our experience progressed in operating to free nerves and vessels from pressure. For the removal of scar pressure not only liberates the fragile axons and restores better blood supply at the point of injury, but it improves the nutrition of the tissues below the wound which had labored under the double disadvantage of insufficient blood and poor trophic stimulation from the spinal centers.

4. *Intraneural Blocking of Axons Without Gross Injury to the Nerve.*—This condition was found in two of our patients. In one of them, following a compound fracture of the bones in the lower third of the leg, in October, 1918, there were, in May, 1919, complete sensory loss in the distribution of the posterior tibial nerve and complete loss of power in the intrinsic muscles of the sole without any signs of regeneration. We had expected to find a complete severed posterior tibial nerve or one badly compressed by scar or callus, but the incision revealed a nerve normal in appearance with only one slight adhesion and without any involvement in scar tissue.

In the other patient there were two gunshot wounds in the arm in the course of the musculospiral nerve, dating from November, 1918. There was a large scar in the axilla and another on the lower half of the arm on its outer and posterior aspect. Pressure on the scars gave pain. Palpation gave no information. The triceps was weak, the supinators and all of the extensors were without function, there was complete reaction of degeneration in the nerve and muscles; there was total anesthesia of the radial cutaneous area. In this case the operation in May, 1919, showed that all the wounds were superficial and the tissues around the nerve in both locations appeared normal, as did the nerve itself.

In each of these patients, therefore, a wound had been received along the course of a nerve, and six to seven months afterward no function had returned in the nerve below the point of injury. Yet there was no change in the nerve trunk, visible to the naked eye, adequate to explain the interruption of function. It appears from these two cases that a trauma which causes no macroscopic change in a nerve trunk can sever its axis cylinders within the epineurium, or at least inhibit their function for six or seven months. We did not have opportunity to study a nerve microscopically to learn what minute changes had occurred. Aside from direct inspection of the nerve after cautious exploratory incision, we know of no diagnostic method by which this intraneural interruption of axons could be differentiated from grosser injuries.

5. *Substitute Movements.*—In studying the voluntary movements of patients the inference is ordinarily justifiable that if a patient is able to pronate his forearm, his pronator muscles and therefore all or part of his median nerve must be sound; if he is able to extend his wrist, that the extensores carpi, and therefore his musculospiral nerve are functioning. Yet care is necessary in making these inferences, for patients in whom the median nerve has been divided with paralysis of the pronator muscles, learn in the course of time to use other muscles to imitate the movement of pronation. Similar substitute movements were observed in other nerve palsies, a few of which we mention (see Figs. 1, 2, 3, 4).

(*a*) An imitation of flexion or extension produced by a pull in the wrong direction by the normal antagonist muscles, and an immediate rebound of the joint. This rebound is easily mistaken for a positive motion, and in these cases it appeared that a muscle actually paralyzed was contracting. Thus, in several patients with complete external popliteal paralysis some dorsal extension of the toes seemed possible. When the toes were carefully watched, the dorsal extension movement was seen to be simply an elastic rebound after a quick and very slight plantar flexion.

(*b*) A paralyzed and fibrosed muscle whose tendon extends over two joints can be pulled on by the action of its antagonists, and thus a movement can be secured which resembles the one formerly caused by the paralyzed muscle. A frequent example of this was the extension of the wrist by patients with musculospiral paralysis through the device of flexing the fingers and thus drawing the extensor tendons taut over the knuckles. This tension on the extensor muscles of the fingers extends the wrist.

(*c*) Patients with complete median nerve paralysis may be able to pronate the forearm by contracting the extensors of the wrist so as to turn the hand through about 90 degrees and then allow the flexed hand to drop by gravity into the prone position. This is easily mistaken for normal pronation.

Fig. 1.—Paralysis of muscles dependent on left musculospiral nerve. In picture A, the patient is trying to extend his wrists while holding the fingers straight. He succeeds in the right hand, fails in the left. In picture B he has extended his left wrist by flexing the fingers. (He unconsciously imitated this movement in the right hand.)

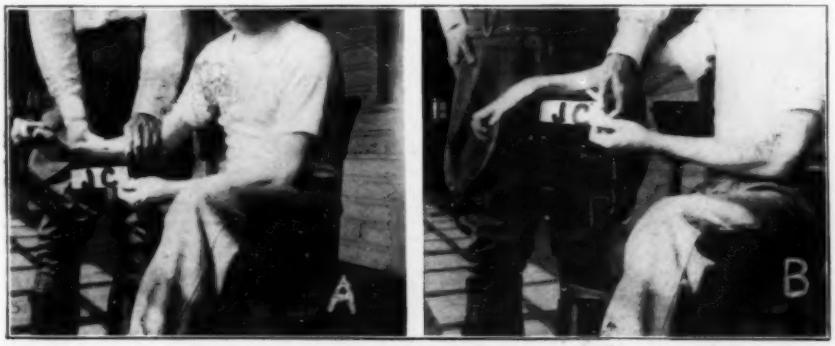

Fig. 2.—Paralysis of muscles supplied by the right median and ulnar nerves; marked contracture of flexor muscles of fingers. In picture B the patient is attempting to flex his fingers beyond the position presented. Picture A shows the amount of flexion secured in the fingers by extending the wrist. It enables him to grasp and hold small objects like a lead pencil.

(*d*) It is believed by some authorities that extension of the second and third phalanges of the fingers is caused only by the interossei (ulnar nerve). But the musculospiral extensors of the fingers also act on these distal joints, as well as on the proximal ones. Thus in ulnar nerve injuries one must not argue that some ulnar function persists simply because extension of the terminal phalanges is possible.

Fig. 3.—Right median nerve injury. Paresis of long flexors of digits. In picture A all fingers are extended. The patient is about to attempt to flex them. In picture B he has flexed his left fingers without disturbing the wrist. In the right hand he flexed the fingers only by extending the wrist.

Fig. 4.—Paralysis of interossei muscles following complete severance of left ulnar nerve. Picture A shows the proximal phalanges held in extension, the second and third phalanges flexed. In picture B the patient has extended the second and third phalanges in spite of the paralysis of the interossei muscles. This was done presumably by the extensor communis digitorum.

II. THE FINER ANATOMICAL STRUCTURE OF THE PERIPHERAL NERVES AS THE ANATOMICAL BASIS FOR NERVE SUTURE; GUIDING PRINCIPLES FOR THE TECHNIC OF NERVE SUTURE AND NERVE GRAFTING

From the beginning of a peripheral nerve operation to its end a perfect technic is necessary. The exposure of the nerve by an incision which will cause the minimum amount of injury to muscles, the freeing of the ends of a divided nerve and the excision of the surrounding scar tissue with the gentlest handling of the nerve, the perfect control of bleeding so as to have a dry wound, the accurate sectioning of nerve bulbs until good nerve fibers are exposed, the proper approximation of the nerve ends without tension, and the properly applied and tightened suture—these and many other details are of great importance. One minute fault in technic, and the chances for a good nerve regeneration are much diminished, no matter how skilfully all other manipulations may have been accomplished.

The ideal technic in nerve suture toward which surgery, therefore, must develop is one that will approximate the proximal stumps of as many as possible of the severed axons to the original glial channels of the peripheral stump through which they formerly reached their destinations; and this must be done without further injurying the delicate axons, and without adding obstacles to their passage into the appropriate channels.

The technic described is the nearest approximation to this ideal we could devise.

1. *The Handling of Nerves During Operation.*—The surgeon cannot be too careful in his handling of the exposed nerves. Great care and gentleness are necessary in all manipulations, and the rule should be to handle the nerves as little as possible. While it is permissible to grasp scar tissue around a nerve with forceps, the nerve should never be held with a steel instrument. One well known operator uses glass hooks for picking up nerves during operation, not because they are softer or smoother than steel, but "for their psychic effect on the operator and assistants." If a nerve is seized by a tissue forceps the fibers thus compressed may be, and no doubt usually are, immediately destroyed at that point. Thus to roughly seize the innervating nerve that goes to the tibialis anticus will cripple the patient the same as if the tendon of that muscle had been cut at the ankle. To nip the ulnar nerve in the arm in a forceps may interrupt the bundle of fibers that control the interossei, and destroy all the finer hand movements.

The nutrition of a nerve is imperiled by dissecting it out of the areolar tissue that surrounds it and supplies it with blood and lymph. There is a strong temptation when operating to "clean up" the nerve

trunk as if for a museum dissection. No further dissection is warranted during operation than that which is necessited to identify the nerve, and to free it from scar pressure. Dissection for identification is required in inverse proportion to the operator's anatomic knowledge.

Furthermore, the lower end of a divided nerve should always be exposed and freed first, because it is the degenerated end. The central end should be exposed for as short a time as possible and should be handled with extreme care. Strong traction should never be made on it in the effort to approximate the divided ends of the nerve. We very emphatically advise against the use of manual methods or of appliances to make rapid or gradual traction on the ends of a nerve. If there is difficulty in drawing together the two stumps, the operator may be tempted to push a finger or blunt instrument along the course of the nerve above and below in order to free it from its surroundings. This dissection breaks away the vascular connections of the nerve, and endangers innervating branches to neighboring muscles. After all inflammatory adhesions and scar bonds have been released, it is doubtful whether the areolar tissue around a nerve restrains it sufficiently to make this procedure necessary.

2. *Salvaging of Nerve Fibers.*—When any bundles of good nerve fibers could be found running through a scar mass, they were carefully dissected out by longitudinal strokes of a knife or blunt dissector, and if possible, saved even though the remainder of the cross section had to be resected and sutured (see Figures 5, 6, 7). But if such a bundle of salvaged nerve fibers lay near the center of the cross section involved it had to be resected to prevent interference with the proper coaptation of the whole nerve.

In many cases where a number of such fasciculi could be discovered in a tangle of fibroneuroma, especially if the record showed that some motor impulses were permeating the obstruction, it was difficult to decide what course to pursue. If extreme conservatism prompted to simply free the nerve from external scar tissue and leave the fibroneuroma unresected, there was the counterbalancing fear that no other fibers but these would ever penetrate the mass, and that even these would sooner or later be compressed and destroyed. On the other hand, there was the fear that when resection and suture had been done, the union would prove unsuccessful and the little function that persisted would thus be lost.

The adage, "leave well enough alone," can always be answered by "good is the worst enemy of best." There are few situations in surgery in which rule-of-thumb is less efficacious, and in which the patient's welfare depends so completely on the sound judgment of the operator, as in this decision whether or not to resect a scar-entangled or frayed nerve.

3. *The Excision of the Bulbous Enlargement or of End Bulbs.*—The bulb or end bulbs should be divided transversely with a sharp scalpel in successive sections until normal nerve bundles without scar tissue around them are exposed. As the upper end of an injured nerve is often swollen, perfectly good funiculi may present edematous or

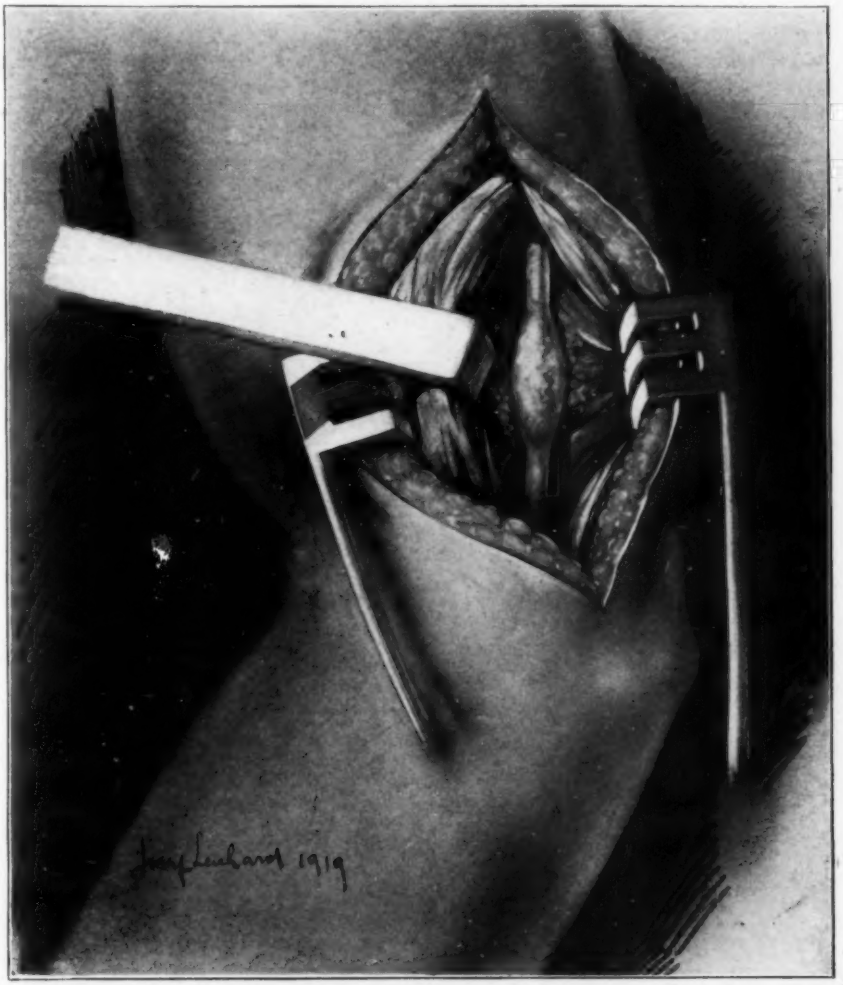

Fig. 5.—Injury of musculospiral nerve by shrapnel; bulb in continuity.

glairy surfaces. There is an interesting and easily understandable difference between the central and peripheral end bulbs (see Figure 8). When the peripheral end bulb is being sectioned the successive cross sections present the appearance of smooth, shiny scar tissue until one section is reached which contains the ends of many normal funiculi

without any scar tissue around them. When, on the other hand, the successive sections of the central bulb are examined, the transition from scar to normal is much more gradual. In successive sections there are an increasing number of good funiculi until an altogether normal transverse section is exposed.

4. *Some Features of the Gross and Minute Anatomy of the Peripheral Nerves, Nerve Pattern, the Prevention of Rotation of the Nerve Ends and Distortion of the Nerve Pattern.*—As we have stated above,

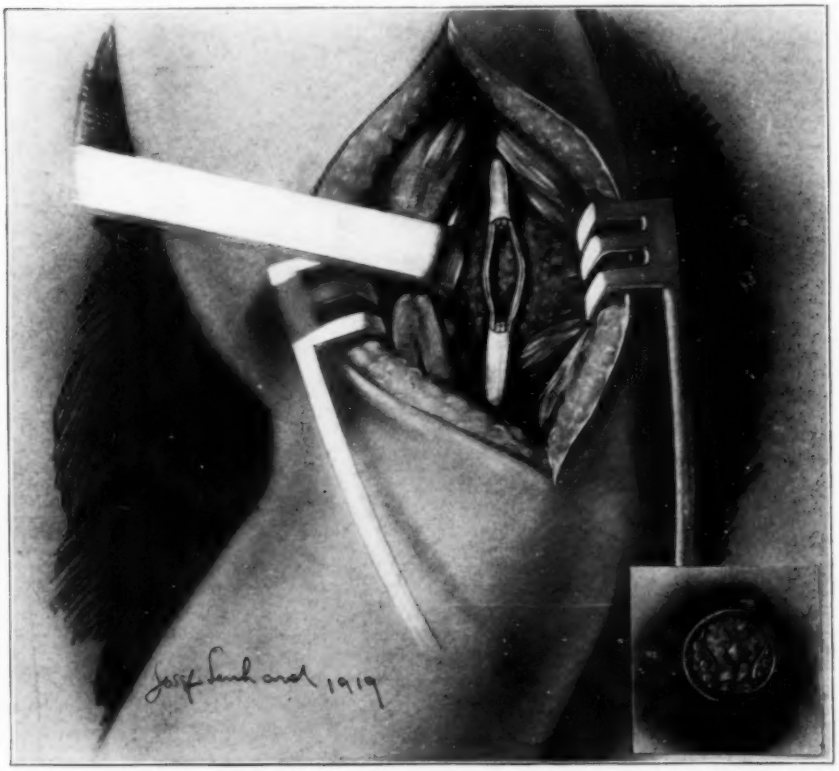

Fig. 6.—Same as Figure 5. The scar tissue has been excised and bundles of good nerve fibers preserved. The insert shows a theoretical transverse section of the bulb.

the ideal apposition of the ends of a nerve would be one in which the cut end of each divided funiculus was placed exactly opposite to its mate. While this ideal cannot be attained, the effort should be made to approach it as nearly as possible. For this purpose, a good understanding of the arrangement of funiculi in the different nerves, and in the different parts of each nerve, is indispensable.

It is remarkable how little is known of this subject. The investigations of Stoffel, Compton, Langley and Hashimoto and others have made it probable that each nerve has a definite nerve pattern, although the pattern may and does vary in different parts of the same nerve. Ideal peripheral nerve surgery can only be done when we have a very complete knowledge of the arrangement of funiculi and fibers in the peripheral nerves. We have made a large number of observations in our nerve operations and have carefully noted the arrangement of funiculi in the cross sections of the nerves. In the majority of

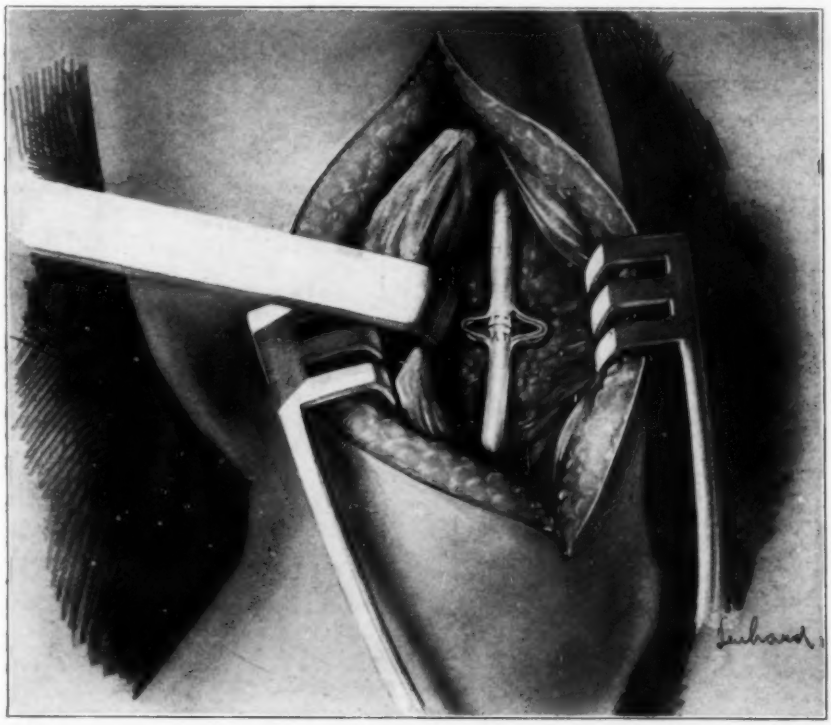

Fig. 7.—Same case as Figures 5 and 6; the suture completed.

instances the nerve patterns in the peripheral and central ends, visible to the naked eye, were either identical or were very similar. An entirely different arrangement in each end was observed in a surprisingly small number of the patients.

This subject is one of such great importance that Dr. Henry A. Riley and one of us (Elsberg) have been investigating it on human cadavers. The studies are far from complete, but they show that in each nerve there is, at different levels, a definite grouping of funiculi, easily recognizable with the naked eye. In many instances these group-

ings are so regular that, by a proper nerve suture, the end of each divided funiculus can be brought into approximate apposition to the corresponding end, and rotation of the ends of the divided nerve and distortion of the nerve pattern can be prevented.

We can obtain additional knowledge of nerve pattern by the electrical stimulation of the ends of a recently divided nerve, and one

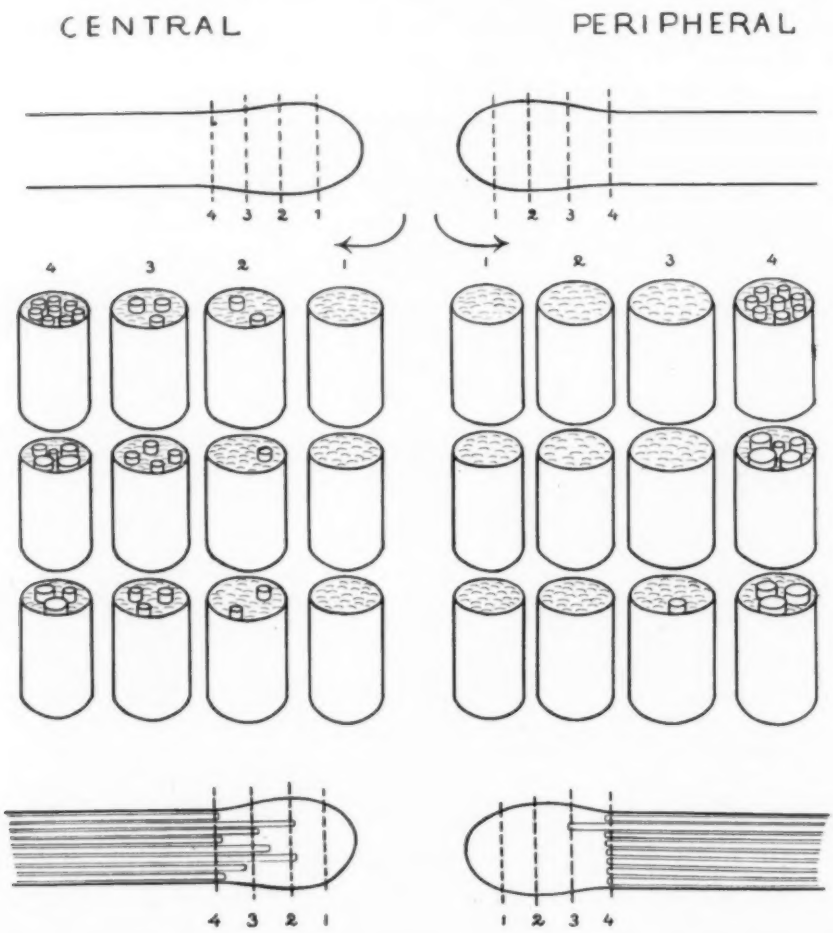

Fig. 8.—The difference between peripheral and central end bulbs.

of us (Elsberg) is also investigating this subject on the nerves of freshly amputated limbs. The investigations are not complete, but a number of definite records have been obtained which we believe are of great importance. A report on these studies will be published later.

The nerves, as regards their funicular pattern, may be divided into two main groups: (*a*) Those in which the number of funiculi is small,

but the funiculi are of large size — the type with large funiculi — and (*b*) those in which the number of funiculi is large, but the funiculi are of small size—the type with small funiculi. The musculospiral and external popliteal nerves belong to the group with large funiculi; the median, ulnar and internal popliteal belong to the group with small funiculi.[2]

Regeneration after suture is more rapid and more complete in the musculospiral and external popliteal nerves which we include in the group with large funiculi than in the median, ulnar or internal popliteal

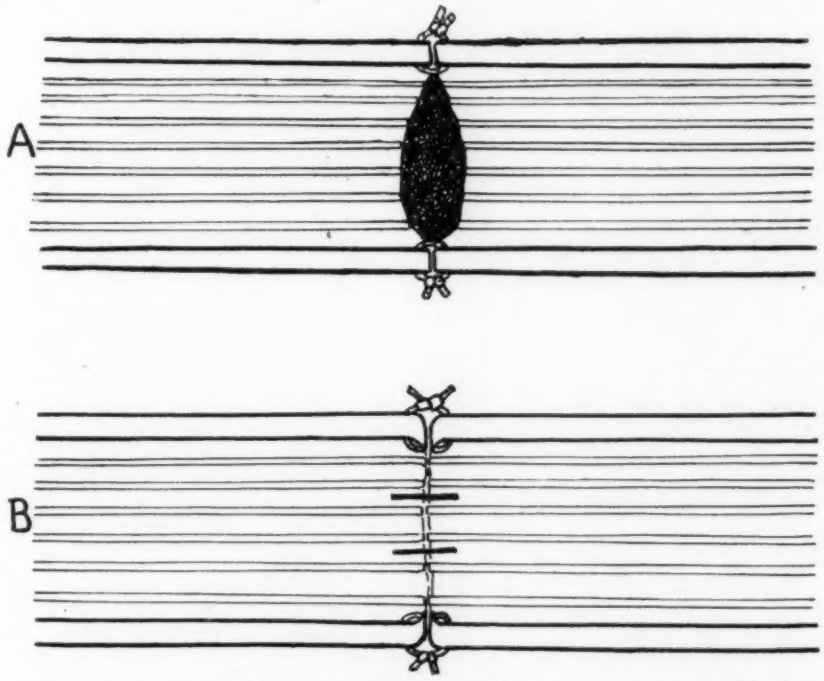

Fig. 9.—A, the condition that may exist when the ends of a divided nerve are united only by epineurial stitches. B, demonstrating more perfect approximation by a combination of perineurial and epineurial stitches (diagrammatic).

nerves with numerous small funiculi. This funicular arrangement may have an important bearing on regeneration after suture, for it is obvious that a given amount of scar tissue is more apt to impede the downgrowth of, and to compress the axis cylinders in a small delicate bundle than in a larger one.

There is another aspect of the subject which is of interest. Severe neuralgias and the so-called causalgia occur most often in the median

2. As we have stated elsewhere, there are variations in the pattern in different parts of the same nerve. This subject will be considered in detail in a separate paper.

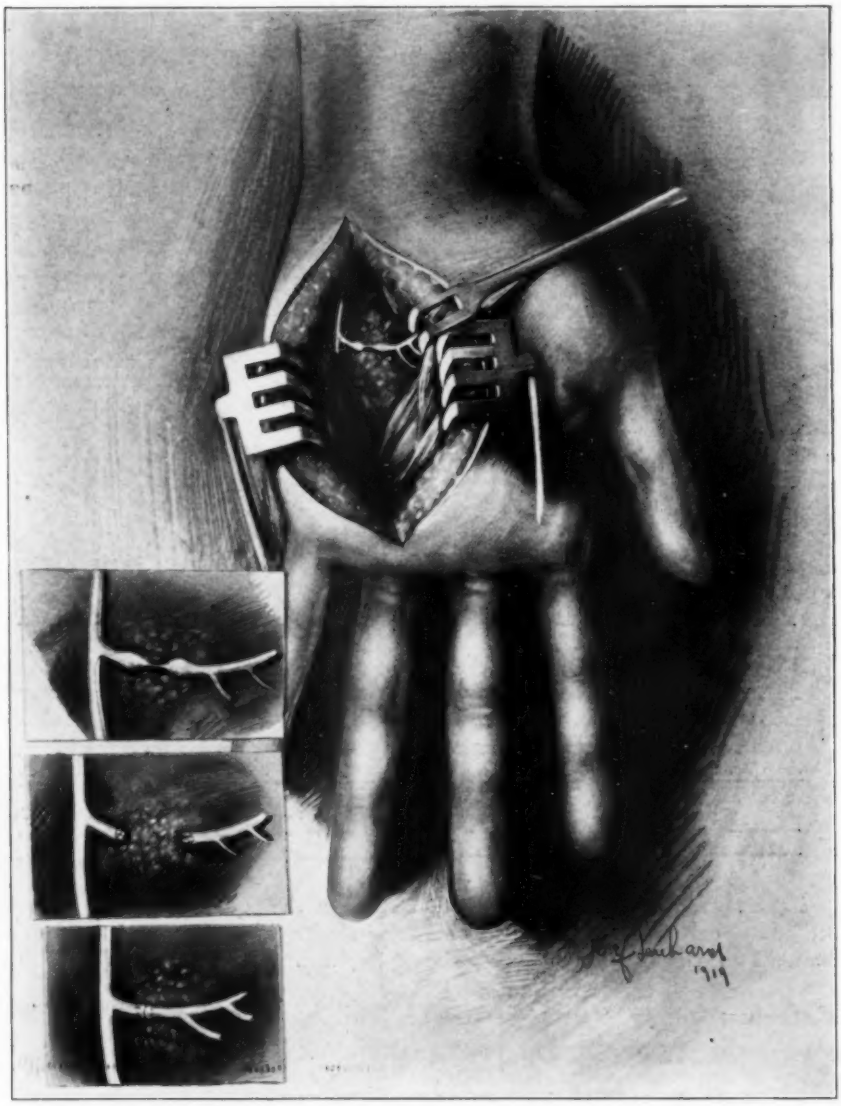

Fig. 10.—Division of deep palmar branch of ulnar nerve by machine-gun bullet. Resection of end bulbs and end to end suture. (The inserts show the successive stages of the operation.)

and internal popliteal nerves with numerous small funiculi. The electrical studies of one of us seem to indicate that in very many instances (perhaps in all) the sensory fibers occupy the most central part of each funiculus. May it not be that in inflammatory processes in the perineurium, an irritation of sensory fibers is much more apt to occur in a nerve with small funiculi than in one with large ones? We hope that the studies we are making will soon permit a definite answer to this question.

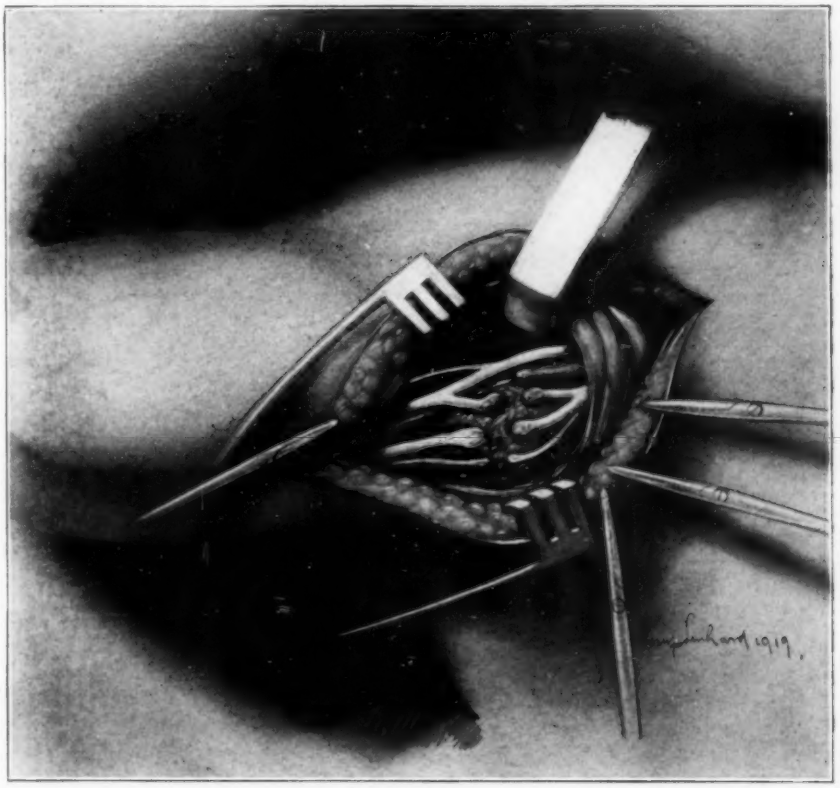

Fig. 11.—Shrapnel injury involving the inner head of the median, the ulnar, the internal cutaneous nerves, and the brachial artery. The end bulbs exposed.

5. *Approximation of the Divided Ends of a Nerve.*—This should always be made without tension. In the majority of instances, this can be accomplished by freeing the nerves—especially the peripheral part—for a considerable distance. In this procedure, however, due consideration should be given to the location of branches and of nourishing blood vessels so that important motor and sensory branches are not injured. The nerve ends can often be approximated without

tension by flexion of adjoining joints (flexion at wrist for injury of ulnar or median nerves in forearm; flexion at elbow for median and musculospiral in arm and forearm; extension at elbow and adduction of arm for ulnar nerve in arm and forearm; plantar flexion at ankle and flexion at knee for posterior tibial; flexion at knee and extension

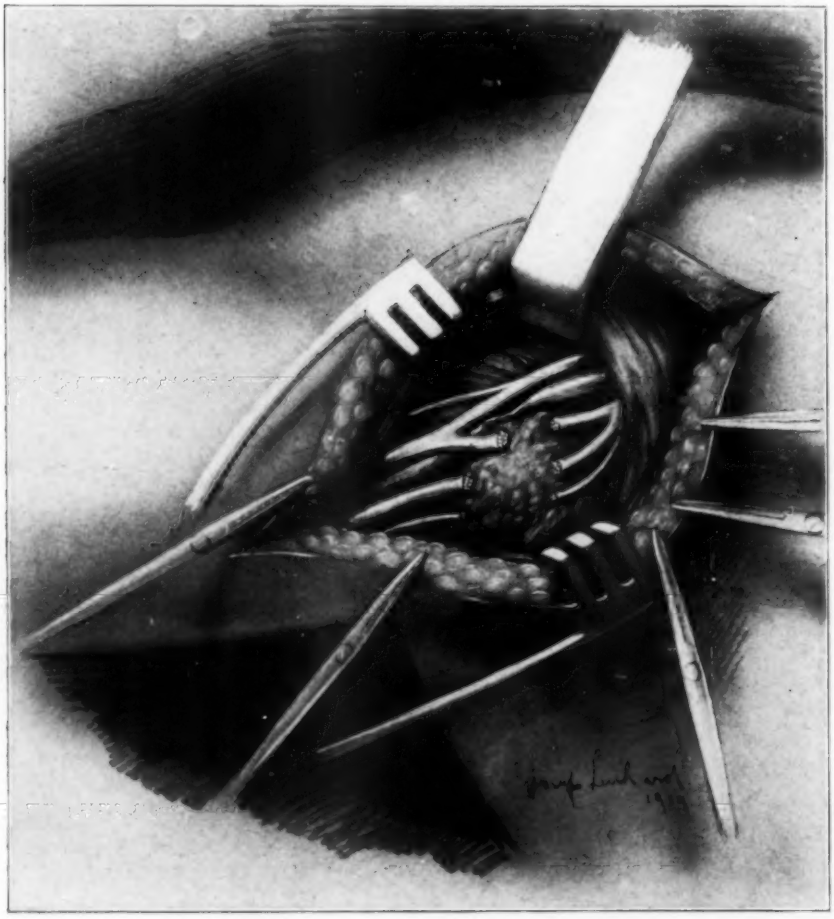

Fig. 12.—The end bulbs resected, showing the funicular pattern of the ends of the nerves.

at hip for sciatic, etc.). Transplantation of the ulnar nerve to the front of the internal epicondyle is often necessary, but in this transplantation the nerve to the flexor carpi ulnaris should not be injured. One of us has made measurements of the amount that can be gained by changing the position of neighboring joints, but this is not the place for a detailed consideration of the subject.

6. *Technical Principles of Nerve Suture.* — We shall not in this paper enter on the finer details of nerve suture, but shall only mention some facts that are essential to this delicate surgical procedure. We believe that for nerve suture the best material is very fine silk (sepa-

Fig. 13.—Shrapnel injury of the posterior interosseous nerve. The end bulbs exposed.

rated strands of the finest silk obtainable) threaded on fine "Carrel" needles. For the actual union, perineurial and epineurial stitches are used. Epineurial sutures alone are not advisable. The funiculi are apt to retract—especially if a little blood or fluid collects between their

ends—and the resulting scar tissue will offer an impediment to the downgrowth of the regenerating axis cylinders from the central end (see Figure 9). After the nerve pattern of each end of the divided nerve has been carefully noted, one, two or three fine stitches are passed through the perineurial tissue — the number depending on the

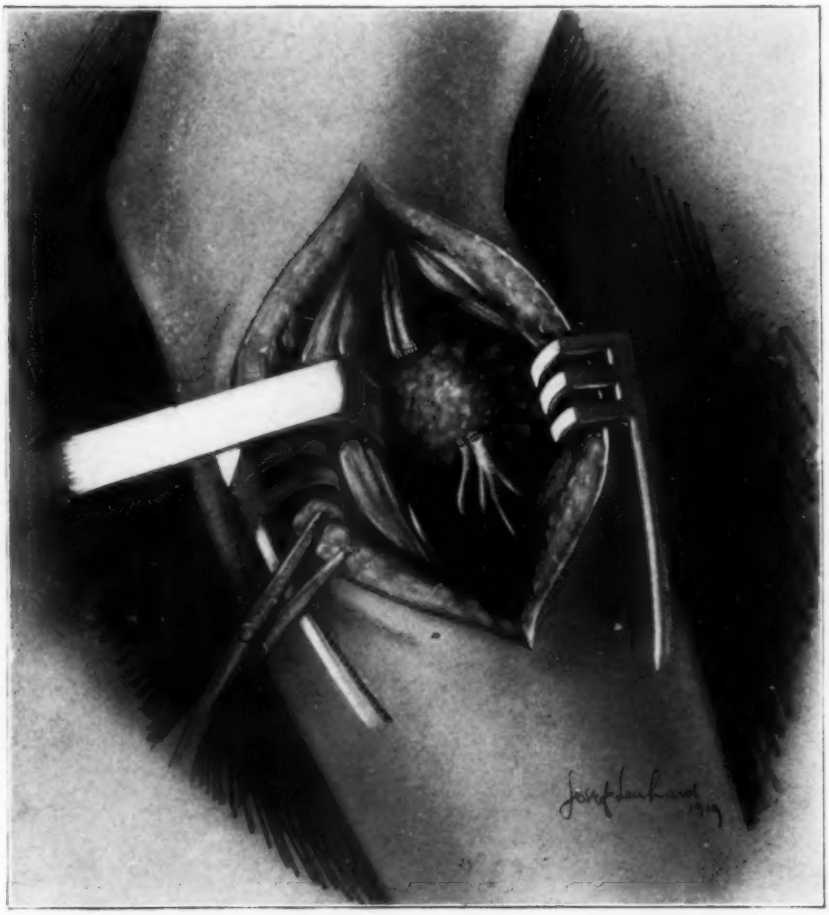

Fig. 14.—Same case as Figure 13. The end bulbs resected, showing the funicular pattern of the ends of the nerves.

arrangement and number of funiculi. No especial difficulty is encountered in passing the stitches through the perineurial tissue between the funiculi or groups of funiculi if the needle and suture material be sufficiently fine. If the sutures are well placed with due regard to the nerve pattern, a fairly accurate apposition of the ends of the funiculi is possible. All of the perineurial sutures should be passed before any

one of them is tied. When the sutures are tied, care should be taken that the funiculi are just brought into apposition. If the sutures are too tightly drawn, the funiculi are bent at their ends with a resulting poor approximation. After the perineurial sutures have been tied, the epineurial stitches must be accurately passed so as to approximate the free borders of the epineurial sheath.

Nerve suture, according to the principles we have outlined, can be done not only on the large nerve trunks, but also on smaller nerves. Thus one of us has been able to recognize the funicular arrangement

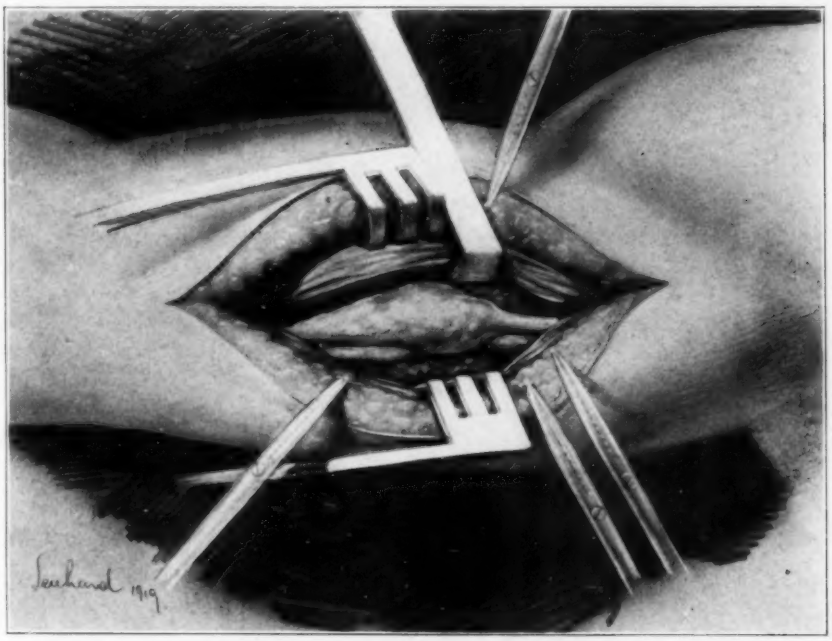

Fig. 15.—Large neuroma following shrapnel wound of median nerve.

and do a perineurial and epineurial suture on the internal cutaneous and lesser internal cutaneous nerves, on the deep palmar branch of the ulnar and on a large muscular branch to the triceps muscle from the musculospiral nerve (see Figure 10, 11, 12, 13, 14).

7. Concerning Procedures When Nerve Suture Cannot Be Done Because the Ends Cannot Be Approximated; Nerve Grafting.—If the condition of a nerve permits it, a neurolysis is always better than a resection and suture, and a resection and end to end suture far better than a resection and grafting.

If by changing the position of neighboring joints or by even moderate tension the ends of the resected nerve could be brought directly together, that was done in our patients in preference to introducing grafts; for the graft is for man as yet an unproved expedient. When the divided ends could not be brought together by reasonable tension and the other expedient elsewhere mentioned, grafts were employed, because nothing else was possible. In such cases the effort was made, whenever feasible, to replace each fasciculus of the main nerve with a fasciculus of the graft (see Figures 15, 16, 17).

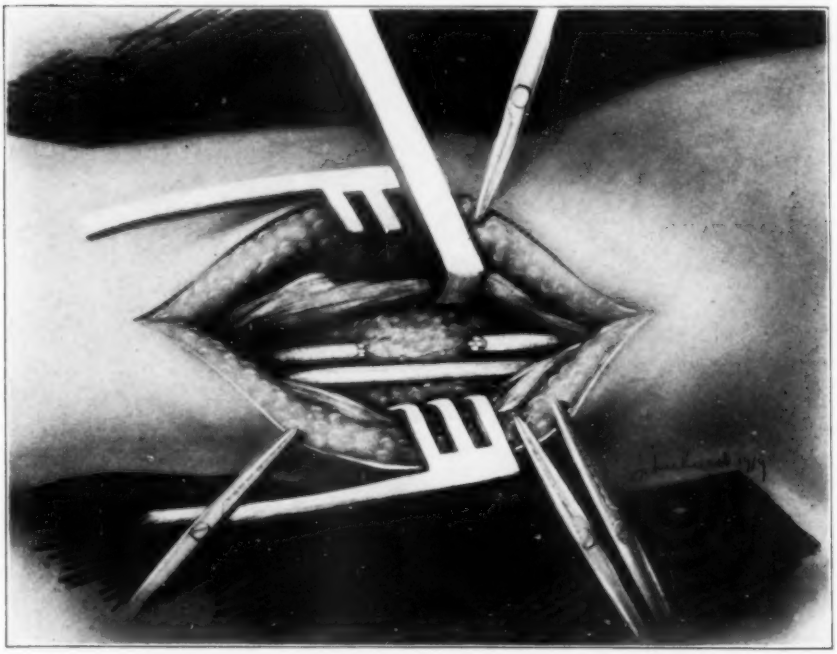

Fig. 16. — Same case as Figure 15. The neuroma has been resected. A defect 5 cm. long remained after the ends of the nerves had been brought as near as possible to each other. Note the funicular pattern.

We shall not enter into a discussion of the comparative value of autografts, homografts and heterografts, and of homografts preserved in cold storage, or in alcohol (Nageotte). Animal experiments have demonstrated that the autograft is to be preferred, and we have limited ourselves to the use of autografts of cutaneous nerves (external cutaneous of thigh; internal saphenous; anterior and posterior cutaneous branches of musculocutaneous; internal cutaneous). These grafts

were obtained and transferred by a method of our own,[3] which was devised in order to minimize the amount of handling of the grafts and to simplify the combination of a number of segments of a cutaneous nerve into the typical "cable graft."

In cases where a graft has been implanted, and after sufficient time has elapsed to allow the new axis cylinders to grow through the length of the graft, it has been recommended that the lower point of union between graft and nerve be resected, freshened and resutured. The

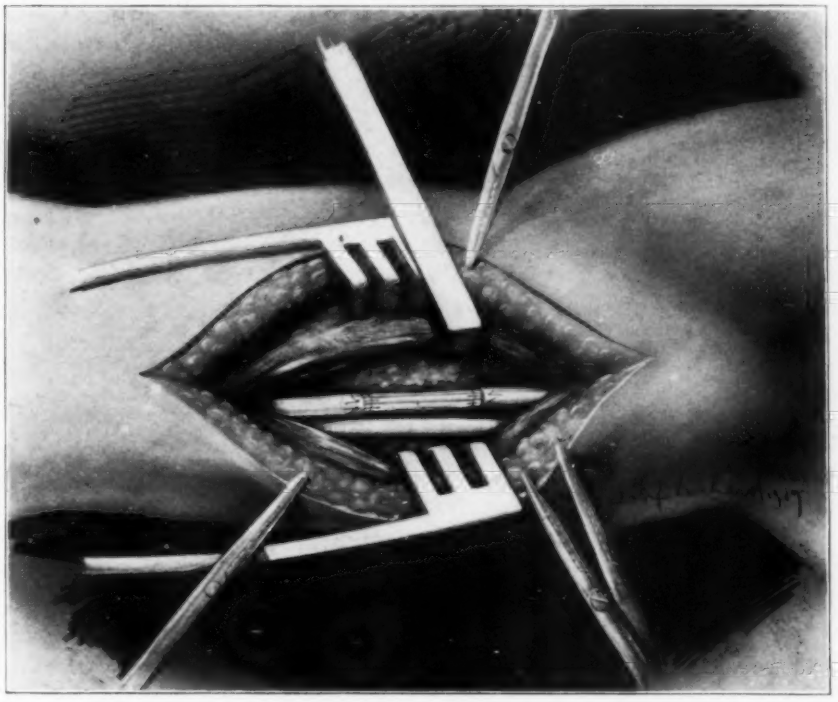

Fig. 17.—Same case as Figures 15 and 16. The cable graft in place. The graft is made up of as many strands as there were fasciculi in the nerve ends.

question here raised is this: Will new axis cylinders penetrate a well established junction better than a fresh one? It is difficult to answer such a question at the present time. One reply seems as reasonable as the other. In the case of one patient whose wound was reopened two months after the graft operation, the lower junction was found to be well established, perfectly soft and difficult to distinguish from neighboring parts of the nerve. It was not molested.

3. Elsberg, C. A.: Technic of Nerve Suture and Nerve Grafting, J. A. M. A. **73**:1422 (Nov. 8) 1919.

III. THE OUTLOOK FOR THE FUTURE

In this paper we have considered only a few aspects of the symptomatology and of the anatomy and surgery of traumatic lesions of the peripheral nerves. The experiences of the world war will soon give us extensive statistics of the results that can be attained. It will be a difficult matter, however, to correctly judge of the end results when they are reported, for the technic of the operation has varied a great deal in the hands of different operators. It will require very careful discriminative studies of the end results when they are reported to determine to what degree the surgical methods used will have influenced the results obtained. We believe that in the advances that have been made, an important part is due to a fuller realization of the need for a better knowledge of the minute anatomy of the peripheral nerves. There can be no queston that, no matter how much nature will help the surgeon, the surgical treatment of injuries to the peripheral nerves must be founded on the effort to bring the ends of a divided nerve into an apposition which approximates as much as possible the relation that existed before the injury occurred. With this aim in view, a very thorough knowledge of the structure of the peripheral nerves, of the course of fibers in the different funiculi, of the number and arrangement of the funiculi and of the functions of the fibers in each bundle is essential. Much attention has been paid to the course of fibers and fiber tracts in the brain and spinal cord, but a knowledge of the course of fibers in the peripheral nerves must still be acquired. The suggestions made in this paper constitute an effort in that direction. We believe that real peripheral nerve surgery will date from such a knowledge, and that the peripheral nerve surgery of the future, based on a minute and detailed knowledge of the finer anatomy and physiology of the nerves and a consequent advance in the finesse and delicacy of technic, will be able to show results far better and more complete than those that have been attained in the past. *"Natura non fecit saltum."*

OVERLAP OF SO-CALLED PROTOPATHIC SENSIBILITY AS SEEN IN PERIPHERAL NERVE LESIONS*

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INTRODUCTION

For many years it has been noted that total loss of sensation after complete division of a peripheral nerve is limited to a much smaller area than we would expect from its anatomic distribution. Likewise, it has been observed that following injury of a peripheral nerve sensory symptoms may rapidly diminish and at times loss of sensation to pin prick be entirely absent.

That severe widespread anesthesia results only from trauma of several nerve trunks of a plexus, has generally been accepted. Lesions of single nerves result in partial anesthesia or, if a severe anesthesia be present, the area of complete loss of sensation rapidly shrinks.

Many attempts have been made to explain these phenomena. Among the older theories were: (1) That nerve fibers grow from healthy surroundings into the insensitive parts;[1] (2) that after section of a nerve stimulation of the severed part may pass through an accessory branch into an adjacent nerve and reach the major branch of the injured nerve above the lesion, through a second lateral branch (collateral innervation);[2] (3) that numerous anastomoses connect the peripheral ramifications of sensory nerves, many cutaneous areas receiving their innervation from different nerves. All these opinions have undergone important changes since the investigations of Head and his co-workers. The results of their brilliant studies led Head, Rivers and Sherren[3] to conclude that "the sensory mechanism in the peripheral nerves consists of three systems:

1. "Deep sensibility, capable of answering to pressure and to movement of parts and even capable of producing pain under the influence of excessive pressure, or when the joint is injured. The fibers, subserving this form of sensation, run mainly with the motor nerves, and are not destroyed by division of all the sensory nerves to the skin.

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*From the Department of Neuro-Surgery, U. S. Army General Hospital No. 28, Fort Sheridan, Ill. Under the direction of Lieut.-Col. Dean D. Lewis.

1. Schuh: Quoted by Oppenheim, Textbook on Nervous Diseases, Chicago, Chicago Medical Book Co. **1**:408, 1911.

2. Letievant: Traite des sectiones nerveuses, Paris, 1873.

3. Head, Rivers and Sherren: Brain **28**:111, 1905.

2. "Protopathic sensibility, capable of responding to painful cutaneous stimuli, and to extremes of heat and cold. This is the great reflex system, producing a rapid, widely diffused response, unaccompanied by any definite appreciation of the locality of the spot stimulated.

3. "Epicritic sensibility, by which we gain the power of cutaneous localization, of discrimination of two points, and of the finer grades of temperature, called cool and warm."

Head and Sherren[4] state that in complete division of a mixed nerve, as the median or ulnar, the area it supplied does not become uniformly insensitive. Whereas previous observers have stated that sensation is diminished over the full area usually assigned to the injured nerve and lost completely over a small portion only, they have shown that this diminution of sensation is in reality a total loss of sensibility to stimulation with cotton wool, to the compass test, to the painless interrupted current, and to degrees of temperatures between 22 C. and 40 C. In this area are felt only the stimuli affecting the protopathic sensibility, such as the prick of a pin and temperatures below 20 C. and above 40 C. The area rendered insensitive to light touch by division of the median or of the ulnar nerve varies little in extent. In sharp contrast to this slight variation is the extreme difference in surface extent of the loss of sensation to a pin prick which follows division of either of these nerves. "The consequence of both division and irritation of these nerves shows that as far as protopathic sensibility is concerned they overlap to an enormous extent."[5]

It is evident, therefore, that the complete sensory distribution of a peripheral nerve consists of its exclusive supply, or that area in which loss of sensation is produced by its division, and in addition its overlap or the area determined by the limits of skin sensitive to stimuli when all the adjacent nerves have been severed. Head and Sherren, employing the method of residual sensibility, were able to determine the complete sensory distribution of some of the nerves. These areas were part of the median, the internal saphenous, part of the external popliteal, the external saphenous and part of the posterior tibial.

The purpose of this paper is: first, to record the smallest area of loss to prick pain which follows interruption of the various peripheral nerves; second, to point out the relative smallness of this area as compared to the area of loss to touch; third, to show that the preservation, or early return of prick pain as compared to tactile sense is due to the assumption of function of adjacent nerves and not to nerve regeneration, as interpreted by Head; and, fourth, to outline the total sensory distribution of some of the peripheral nerves by residual sensibility.

4. Head and Sherren: Brain **28**:283, 1905.
5. Head and Sherren: Brain **28**:295, 1905.

MATERIAL

My observations were made on 500 patients with peripheral nerve lesions seen early in base hospitals in France, and 520 patients with peripheral nerve lesions studied later at U. S. Army General Hospital No. 28, Fort Sheridan, Ill.

The observations of early peripheral nerve lesions were in most instances uncontrolled by operative procedures. A large proportion of the lesions were partial and frequently complicated by injuries to adjacent small sensory branches. But these observations served a useful purpose. They showed: (1) that in many cases for the first two or three weeks only a very small area within the border of the part insensitive to cotton wool was sensitive to pin prick; (2) that in a few a larger zone sensitive to pin prick appeared within fifteen days; and (3) that the return of sensitiveness to pin prick in a larger zone, corresponding to the area which we later determined as overlap, usually was found, at times variable from thirty to one hundred days. The cases showing return to pin prick over a large area in less than thirty days were predominantly cases of radial and musculospiral lesions.

The material of peripheral nerve lesions studied later may be divided into two groups: the first, a group of 391 cases of peripheral nerve lesions uncontrolled by operation, and in the majority of instances recovering spontaneously; the second, a group of 129 cases controlled by operation.

CASES CONTROLLED BY OPERATION

Nerve Lesions	Number
Ulnar	20
Radial	31
Median	9
Ulnar and median	15
Median and radial	2
Musculocutaneous, ulnar and median	2
Brachial plexus	2
Great sciatic	20
External popliteal	25
Anterior tibial	2
Ulnar, median and internal cutaneous	1
Total	129

My general impressions relative to the sensory changes in peripheral nerve lesions were derived from the whole material. The areas of total nerve supply and of overlap were obtained only from cases certified by operation. The cases which have been used in the study of regeneration of nerves likewise were certified by operation.

Therefore, although the whole 1,020 cases contributed to my general conclusions concerning these problems, only one group, consisting of the cases coming to operation, was employed in obtaining the data which serve as the basis for the special conclusions contained in this article.

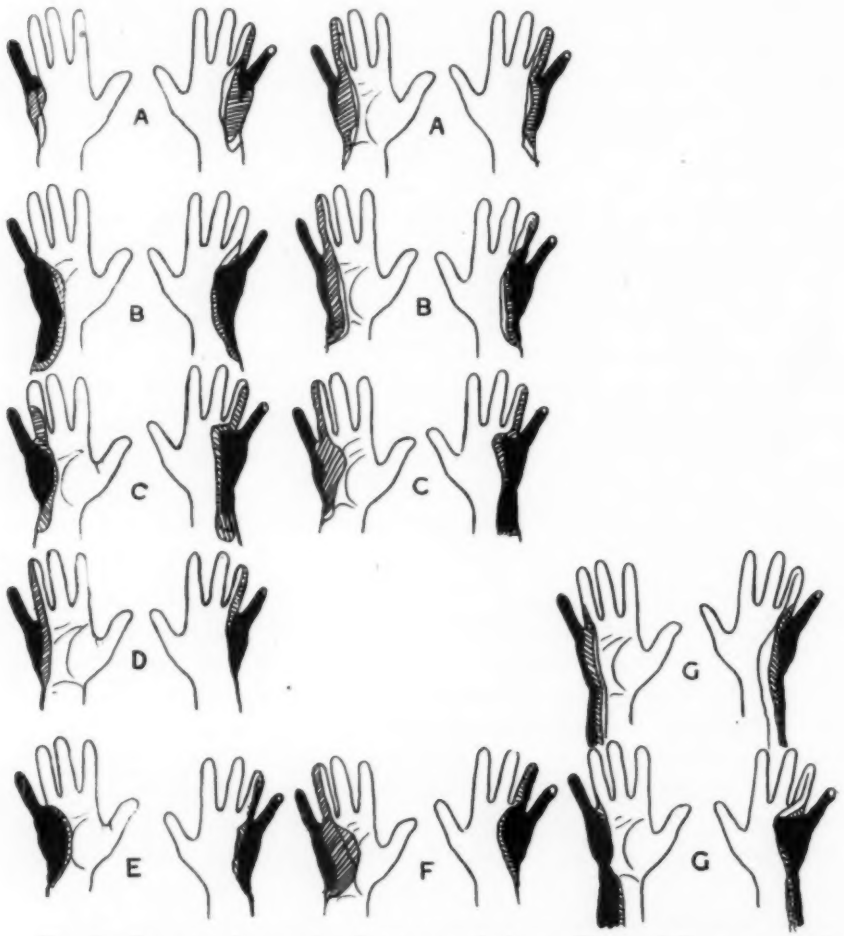

Fig. 1.—Sensory changes in ulnar nerve lesions: Diagonal lines, anesthetic to touch; black lines, loss of prick pain and touch sense; continuous line, borders of loss of temperature sense. The same scheme of charting is followed in all of the diagrams. Where duplication of letters occurs the first is preoperative and the second postoperative sensory chart.

METHODS OF INVESTIGATION

The problems under investigation were studied from a clinical and not from a psychophysical standpoint. The areas of overlap were found in the course of clinical examinations of a large group of cases. The methods of examination, therefore, were those ordinarily used

clinically. The sense of touch was tested by a wisp of cotton. The sensation of pain in response to the prick of a pin was ascertained by using a weighed needle sliding within a bit of glass tubing, so that with different weighed needles a pressure of from 5 to 35 gm. could be applied.

Although in this report temperature sense will not be referred to because of difficulty of standardizing methods and the impossibility of employing the finer methods clinically, it may be stated that for the rough examination of sense of cold, we used a pledget of cotton twisted to a point and saturated with ether. Ether has been employed in the form of droplets by von Frey. We have found that our method per-

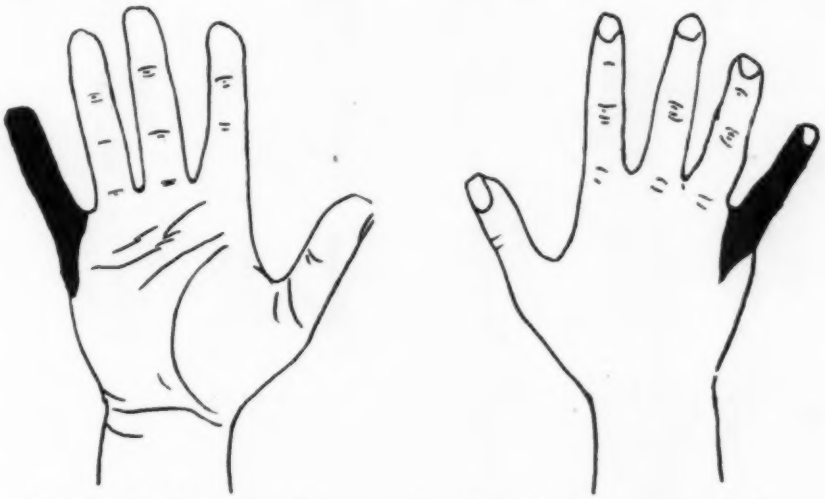

Fig. 2.—Smallest composite area of analgesia in ulnar nerve lesions.

mits a less diffuse type of stimulation and has the advantage of ease and simplicity. For physiologic research this method is, of course, inapplicable and therefore our results will not be incorporated in this discussion.

It must be determined whether our methods are acceptable as to the differentiation of so-called epicritic and protopathic senses and deep sensibility. Light touch with a wisp of cotton to determine sense of touch may, I think, be accepted if exact threshold of sensation is not under investigation, and if exact borders of loss of sense of touch be not insisted on. For the purposes of this investigation, the exact borders of loss of sense of touch need not be insisted on. Only one factor must be considered in this method of examination, namely, return of so-called hair sensibility must not be confused with touch; hence, in testing for touch where an accurate border was to be determined, the parts were closely shaven.

The degree of pressure which it is permissible to employ in determining prick pain without jeopardizing the results by confusion with pressure pain remains to be discussed. Although, as pointed out by Head and Sherren,[6] deep sensibility may be evoked when testing for touch with a stiff roll of wool, this objection is not valid for determining prick pain, within certain limits. A sharp needle was used by Head and Sherren in their early clinical investigations, care being taken to

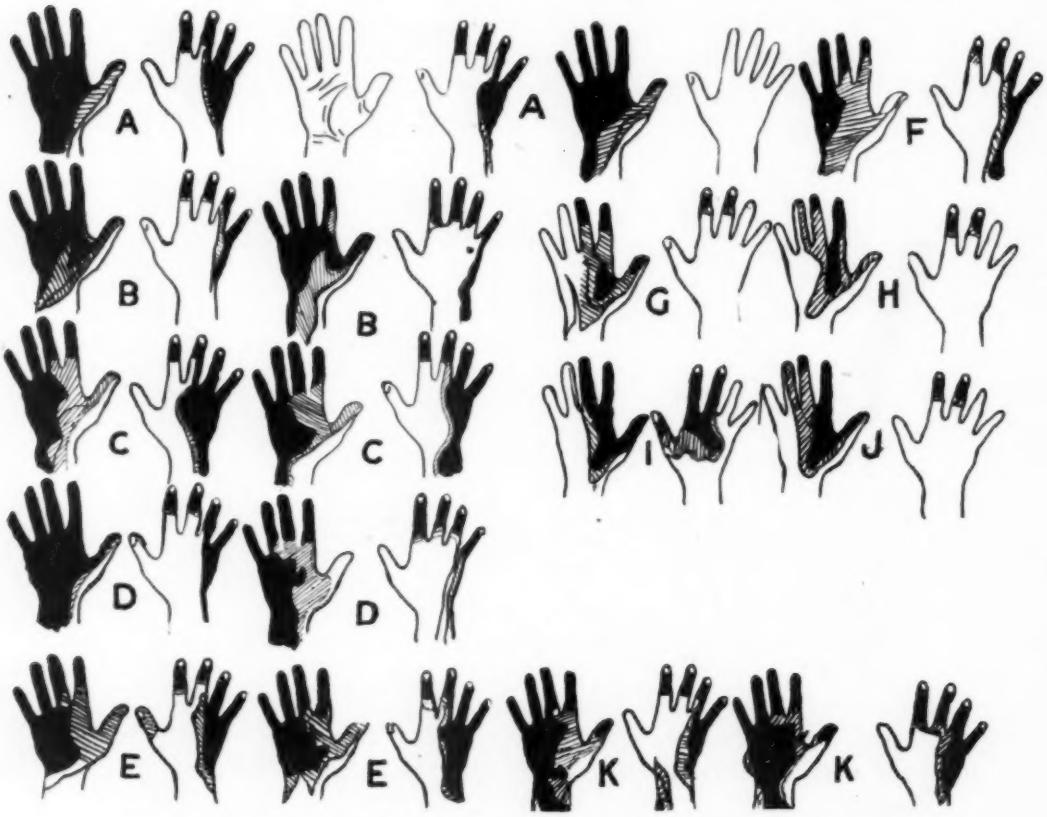

Fig. 3.—Sensory changes in median nerve lesions: *g, h, j,* radial and median; *i,* ulnar and median; *b, e,* ulnar, median and internal cutaneous; *a, c, d, e,* ulnar, median, and musculocutaneous, *k.*

differentiate between sense of deep pressure and true pain. Boring[7] says that "In determining the pain threshold it was especially necessary not to exceed pressures of 5 gm. Although at high intensities of stimulus the introspective difficulty of abstracting from pressure was less with pain than with cutaneous pressure, the greater intensities

6. Head and Sherren: Brain **28**:120, 1905.
7. Boring: Quart. J. Exper. Physiol. **10**:16, 1916.

frequently drew blood and therefore were abandoned." As in Boring's work it was necessary to examine a small area of skin repeatedly and at very short intervals for all forms of sensation, his objection is valid. In my case, on the other hand, it was necessary only to examine sense of prick pain in areas of overlap and not to confuse this pain with pressure pain. We have never found pain to result from 35 gm. of pressure with a blunt object, and care being taken to obtain from the patient responses only to pain from prick of a sharp point, I believe pressure of even 35 gm. to be permissible to map out the overlap of sense of prick pain. No exact measurements of threshold to prick pain were made and in the majority of cases pressure did not exceed 30 gm.

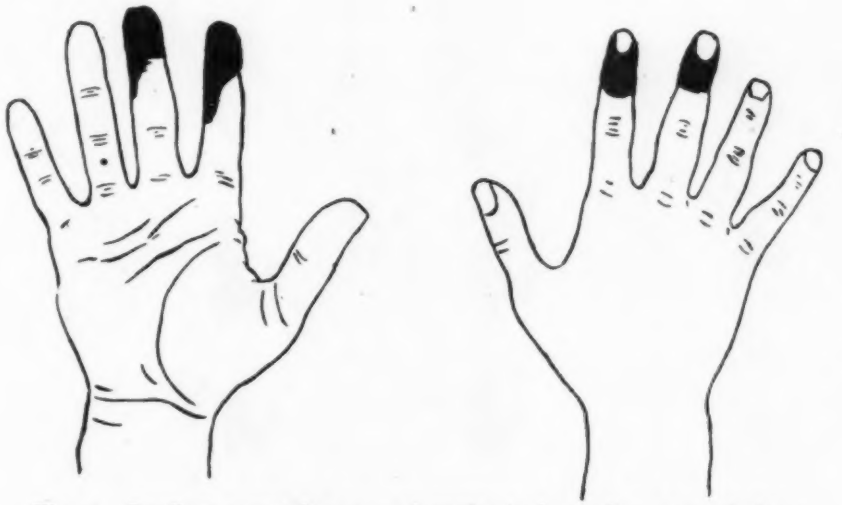

Fig. 4.—Smallest composite area of analgesia in median nerve lesions.

1. EXCLUSIVE NERVE SUPPLY

As I recognized that following section of a mixed nerve the loss to prick pain occupies an area much smaller than the loss to touch, the first part of my task was to ascertain the smallest area which is insensitive to pin prick following section of various nerves. This would indicate the limits of any possible overlap.

Although only a small portion of the area insensitive to touch is quite insensitive to pin prick, diminution of pain sense is present in a large part of the area insensitive to touch, and if graduated degrees of pressure be employed, concentric rings of analgesia are demonstrated. However, we are concerned not with the question of whether any hypalgesia is present, but whether any portion of the skin in the recognized anatomic sensory distribution of a nerve is at all sensitive to pain, provided this pain be due to superficial sensibility. If a part of

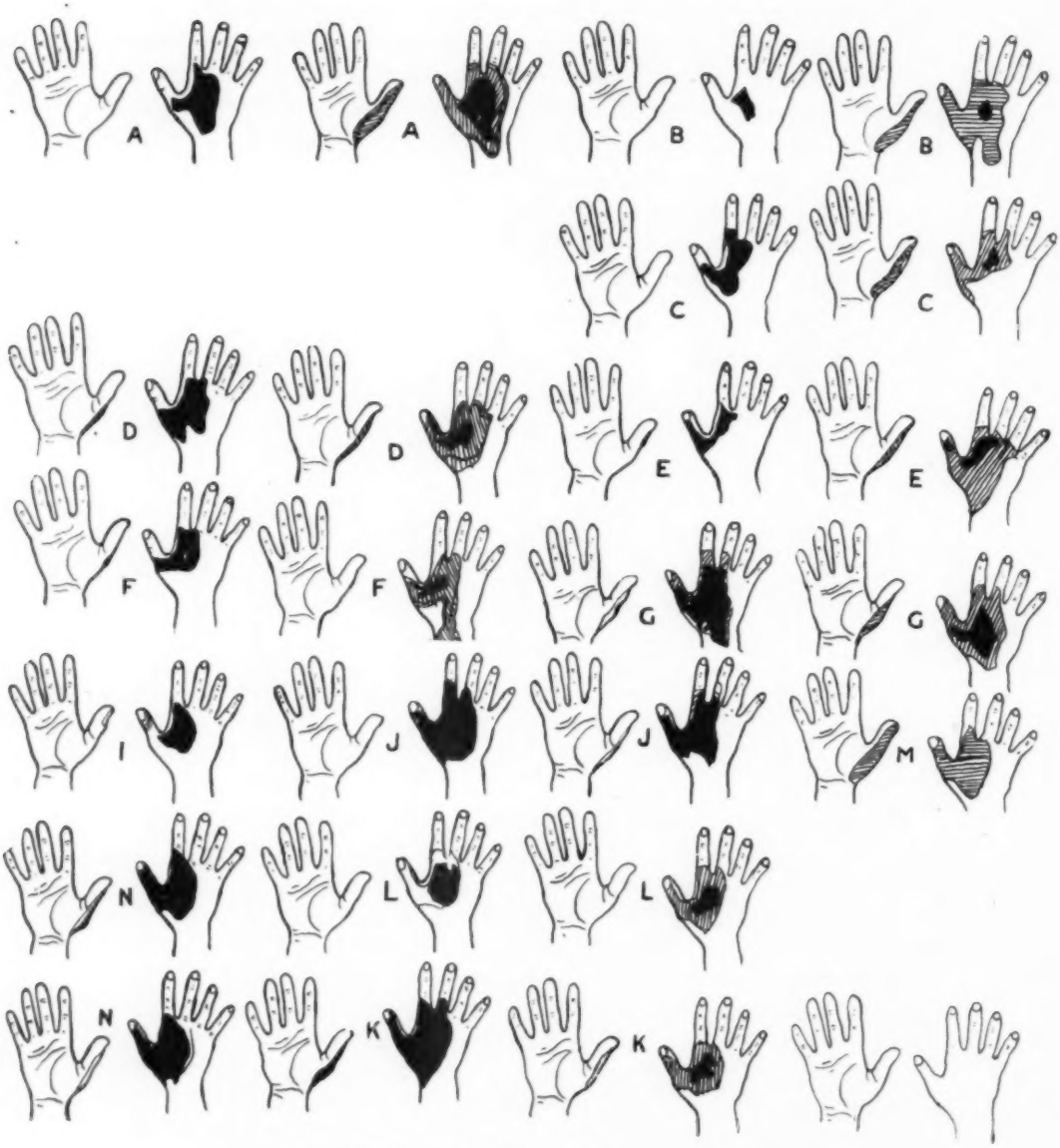

Fig. 5.—Sensory changes in radial nerve lesions.

the skin is sensitive to pain, when a nerve is divided, this sensation must be derived from some source other than this nerve.

To delineate the area exclusively supplied with pain sense by a given nerve one of two conditions must be present: First, the presence of pain sense having been demonstrated within the area of a nerve's supposed anatomic supply, that nerve is found at operation to be divided and the ends separated. Second, the nerve having been seen to be divided, presence of pain sense is demonstrated in its distribution

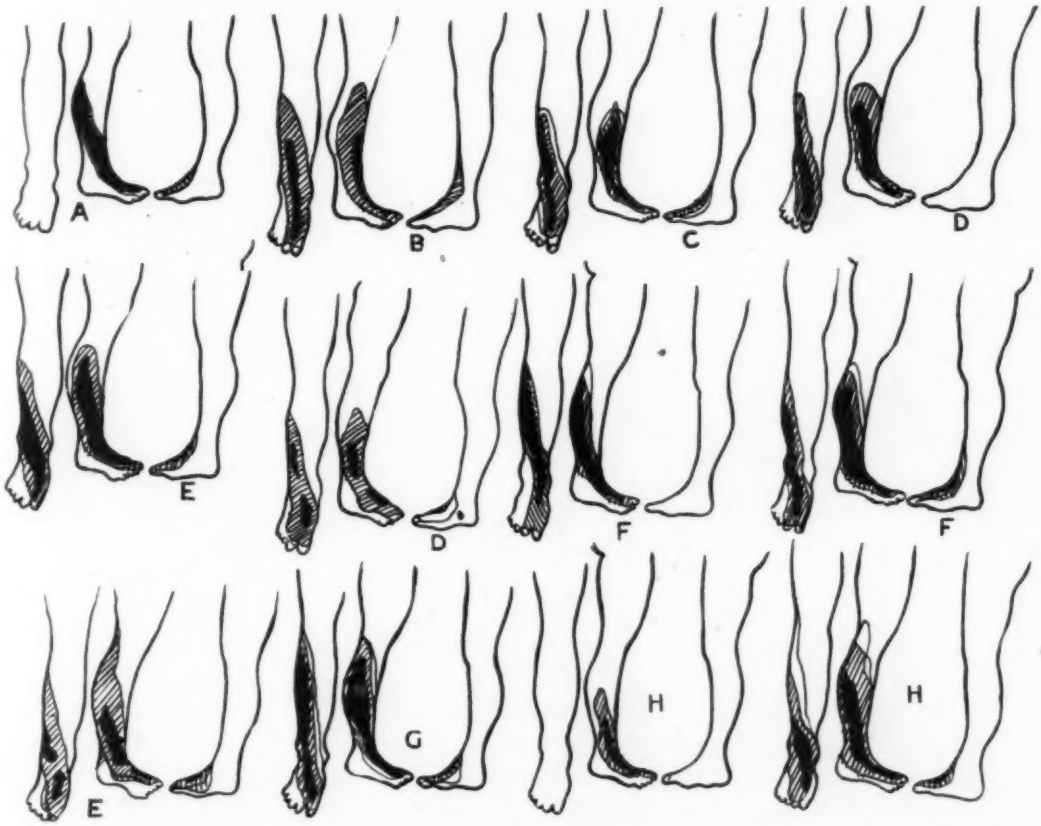

Fig. 6.—Sensory changes in external popliteal lesions.

within the length of time given for the return of protopathic sensibility (Head, Rivers and Sherren, forty-three days). In my cases, under the second condition twenty-eight days was the limit, with the exception of the radial nerve in which the limit was thirty-seven days.

The relatively few cases studied does not make it profitable to attempt to outline the exclusive supply of peripheral nerves to both epicritic and protopathic sensibilities. Suffice it to say that our results

as to the nerves in the hand are in general accord with Stopford,[8] who found in the ulnar nerve some variation from the accepted area of epicritic sense in 20 per cent. of the cases and in the median nerve, in 38 per cent. In three of my cases of median nerve section anesthesia was present over the dorsal surface of the distal phalanx of the thumb.

In ulnar nerve lesions superimposing the outlines of complete analgesia, in the cases shown in Figure 1, *a, b, c, d, e, f* and *g*, the smallest area of analgesia was found to occupy the palmar and dorsal surfaces of the little finger, extending over the dorsal surface of the hand in a triangular area over the fifth metacarpal bone to one-third of its length (Fig. 2). The area included between the borders of the accepted sup-

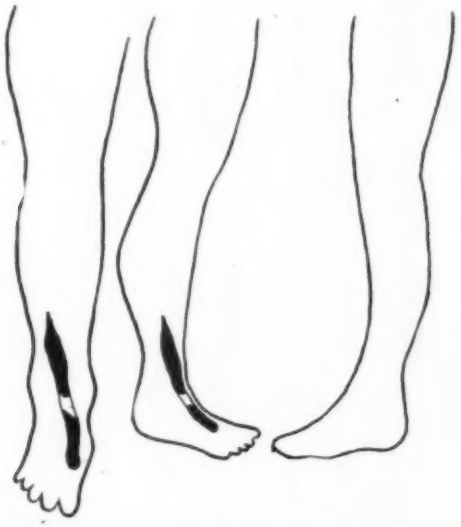

Fig. 7.—Smallest composite area of analgesia in external popliteal lesions.

ply of the ulnar nerve and the borders of this analgesia represents the possible supply of overlapping nerves to pain sense.

The inner border of the smallest area of exclusive supply to pain of the median nerve was obtained in the same way from cases in which the median nerve was subsequently found to be divided (Figure 3, *g, h, i, j*). The outer border was obtained from these cases and in addition from cases of combined ulnar and median lesions which at operation were likewise found to be anatomic divisions, with the ends separated (Fig. 3, *a, b, c, d, e, f, k*). The exclusive supply of the median nerve to pain sense was found to occupy the dorsal and palmar surfaces of the distal phalanges of the index and middle fingers, the ulnar half of the palmar surface of the second phalanx of the index

8. Stopford: J. Anat. **53**:14 (October) 1918.

finger, part of the ulnar portion of the distal half of the second phalanx of the middle finger and the dorsal surface of less than half of the second phalanges of the index and middle fingers. Despite the fact that this small area of total analgesia in median nerve lesions has been recognized,[9] it is necessary at this point to call special attention to this observation as from the study of this nerve much of our evidence relative to overlap was obtained (Fig. 4).

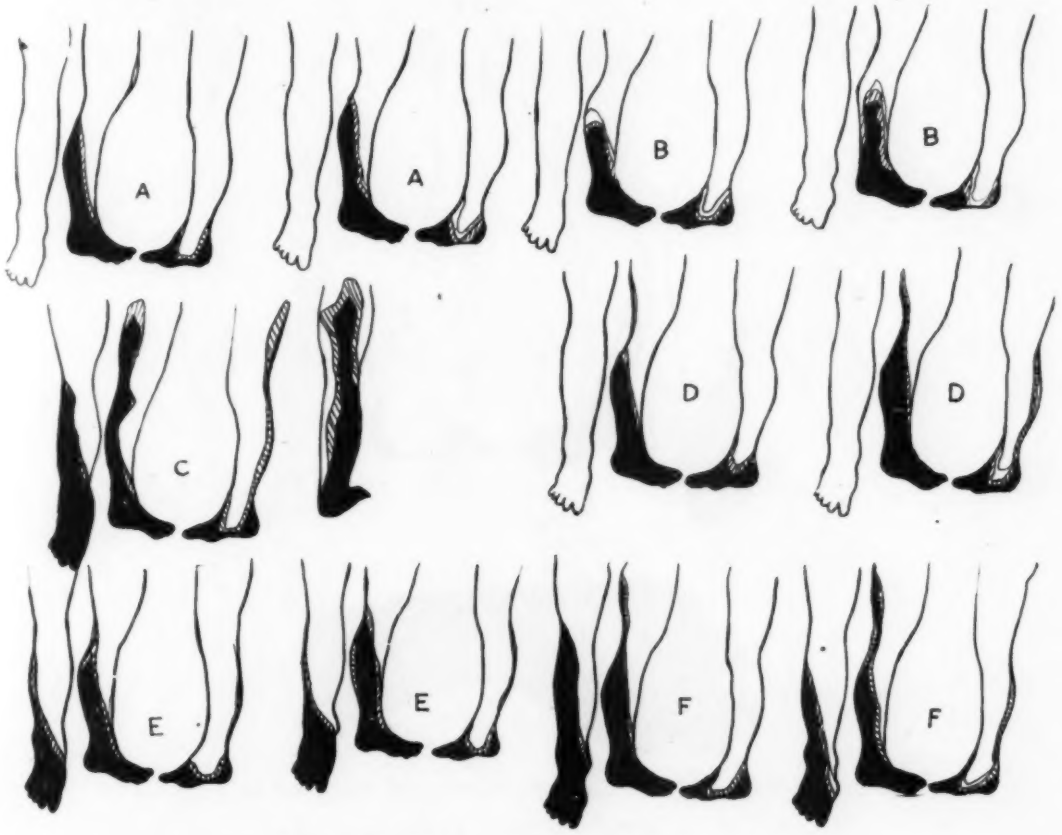

Fig. 8.—Sensory changes in sciatic nerve lesions.

The cases of radial nerve lesions, certified at operation or examined less than thirty-seven days after resection and suture, showed a wide variety of areas of analgesia and in one case no analgesia at all. (Fig. 5, *a* to *m*). Head and Sherren's case 44 of division of the radial nerve where it passes under the tendon of the supinator longus, likewise showed no loss to prick pain.

9. Benisty, Athanassio: Clinical Forms of Nerve Lesions, London, 1918; Head and Sherren: Brain **28**:136, 1905.

Although not infrequently recorded, we have not observed any case of radial nerve lesion which did not show loss of sensation to touch. Of all the peripheral nerves, the radial shows the greatest variation in the areas of loss of sensation to both epicritic and protopathic sensation. This is due to the fact that six nerves are concerned with the sensory supply of the dorsum of the hand; the median, radial, antibrachii posterior branch of the musculospiral, musculocutaneous and ulnar.

Fig. 9.—Smallest composite area of analgesia in sciatic nerve lesions.

Stopford[8] emphasized, as did Head and Sherren, the importance of the musculocutaneous nerve in the supply of the dorsum of the hand, and states that its terminal branches may extend on to the dorsum of the metacarpus, and "it appears that the extent of its distribution varies inversely with that of the radial." Although this may be true, it must not be forgotten that the median nerve must be considered in the supply of sensation to the dorsal area over the distal portion of the metacarpus and the distal portion of the thumb.

One of the reasons for varying reports relative to the sensory loss in radial nerve lesions is the hairy nature of the area of skin under

investigation. The early return of hair sensibility frequently is confused with the presence of sense of touch. The skin must be closely shaven in all cases where examination of touch sense is contemplated. In our charts, where touch is outlined, this precaution was taken. Where examinations were made in the presence of hair, areas sensitive to "touch" frequently were analgesic.

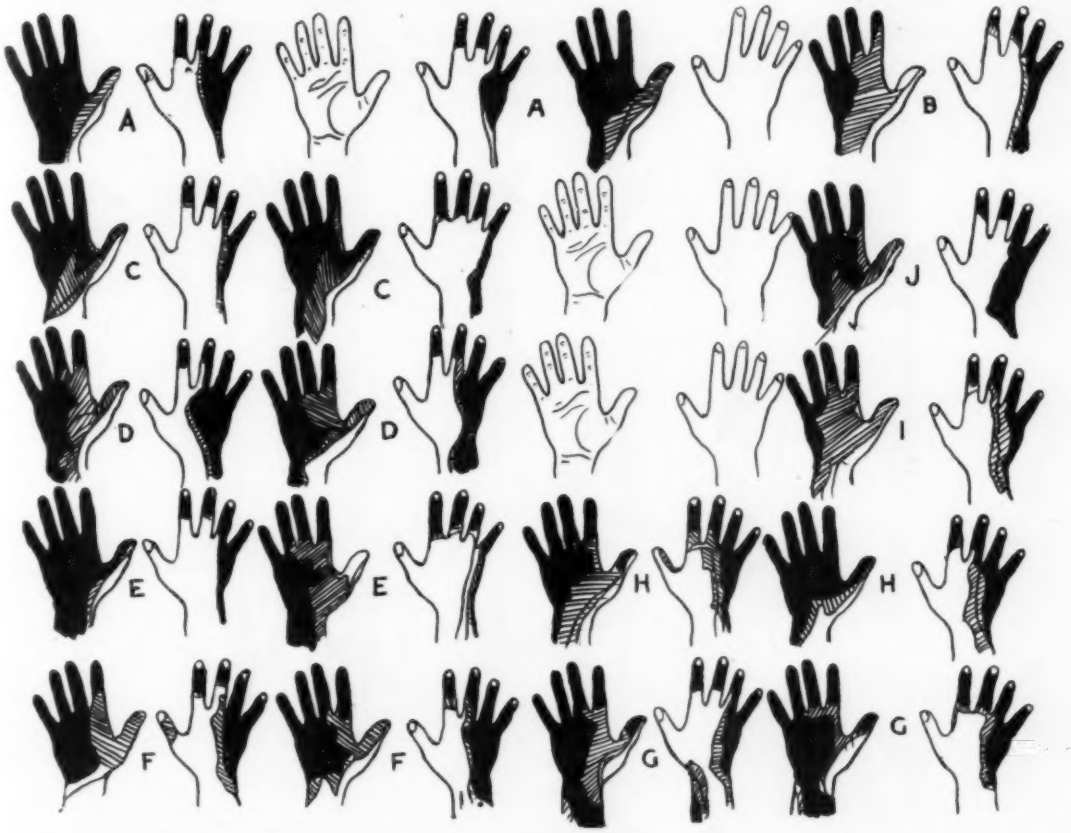

Fig. 10.—Sensory changes in combined lesions of the ulnar, median and internal cutaneous nerves.

No area of skin is exclusively supplied by the radial nerve for prick pain.

The area of exclusive supply of pain of the external popliteal nerve was obtained from certified cases of division and cases examined less than thirty-seven days following resection and suture (Fig. 6, *a* to *h*).

The area consists of a narrow band extending from a point a little above the junction of the lower and middle one-third of the outer surface of the leg, diagonally across the dorsum of the foot to a point

over the middle of the metatarsal bone of the great toe. It is interrupted at the junction of its lower and middle one-third by an area which is sensitive to pin prick. The area is due to the overlap on one side of the internal saphenous nerve and on the other side, the internal popliteal nerve. Although a number of cases showing such an interruption in the band of analgesia have been observed, they have not fulfilled the requirements which we demanded in estimating exclusive supply. One case, Figure 6, *e*, showed this type of interruption of the band of analgesia twenty-seven days after resection and

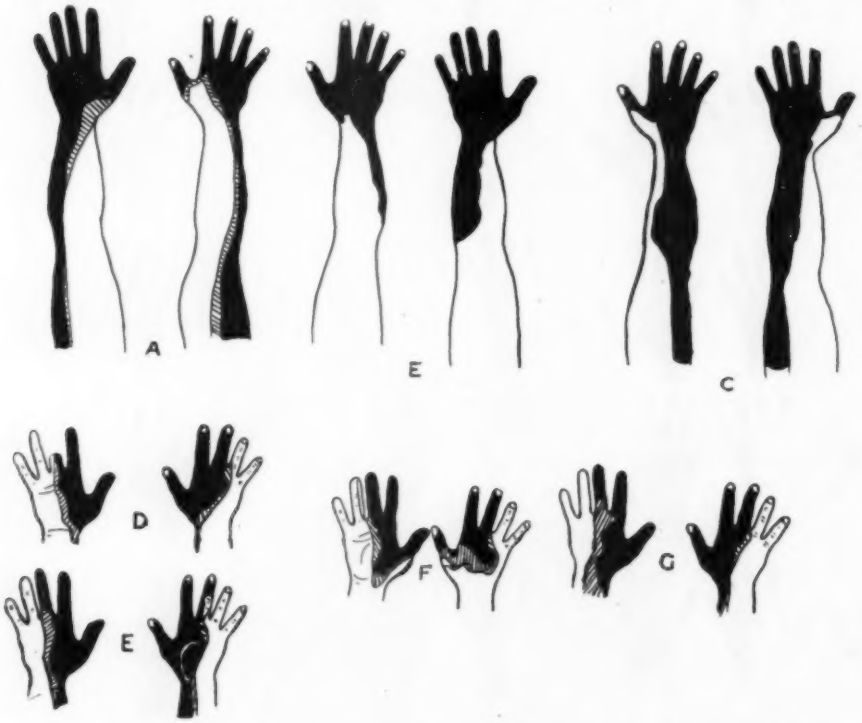

Fig. 11.—Sensory changes in combined lesions of the ulnar, radial and median nerves, *a, b, c,* and of the median and radial, *d, e, f, g.*

suture. Another case which was examined fifty-three days after resection and suture is shown in Figure 6, *d,* but was not used in estimating the isolated supply.

Our results, therefore, are at variance with Head, Rivers and Sherren,[3] who, referring to the fact that the unit for protopathic sense is the posterior root, said that following section, the external popliteal, representing a large part of the fifth lumbar root, does not show a great difference between the borders of the areas of analgesia and anesthesia.

The external popliteal nerve has a surprisingly small exclusive area of pain sense (Fig. 7).

The area of the sciatic nerve was obtained from cases certified to be anatomic divisions, Figure 8, *a* to *f*. This area is illustrated in Figure 9 and need not be described.

Inasmuch as the results above illustrated represent the smallest area of exclusive supply of various nerves for pain, it is necessary to define to what extent they may be used in formulating our ideas

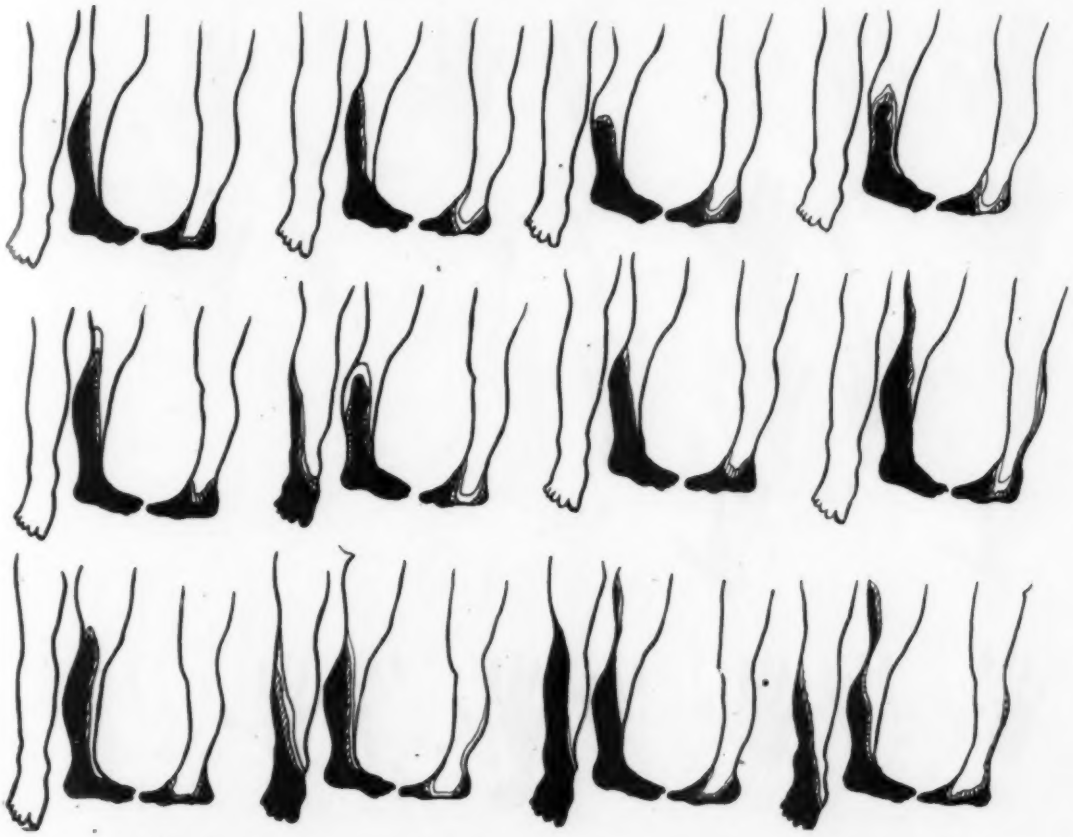

Fig. 12.—Sensory changes in combined lesions of internal and external popliteal portions of sciatic nerve.

relative to nerve overlap. I recognize that in some instances such small areas may be present only when we are dealing with the group of 25 per cent. of cases showing unusual distribution of sensory nerves. These areas are used, therefore, only in establishing a certain limit beyond which it is not permitted to go in interpreting return of sensation to pain as a sign of nerve regeneration. Any return of sense of pain in regions without these borders may be due to unusual nerve

distribution or sensory overlap, and represents possible areas of overlap. It will be found that the areas of overlap I will later describe are not as extensive as these areas would permit us to assume were we to use exclusive pain sensibility as an indication of the borders of overlap.

2. NERVE OVERLAP

I maintain that the return of sensibility to pin prick, which occurs before the return of sensibility to touch, occurs in regions which

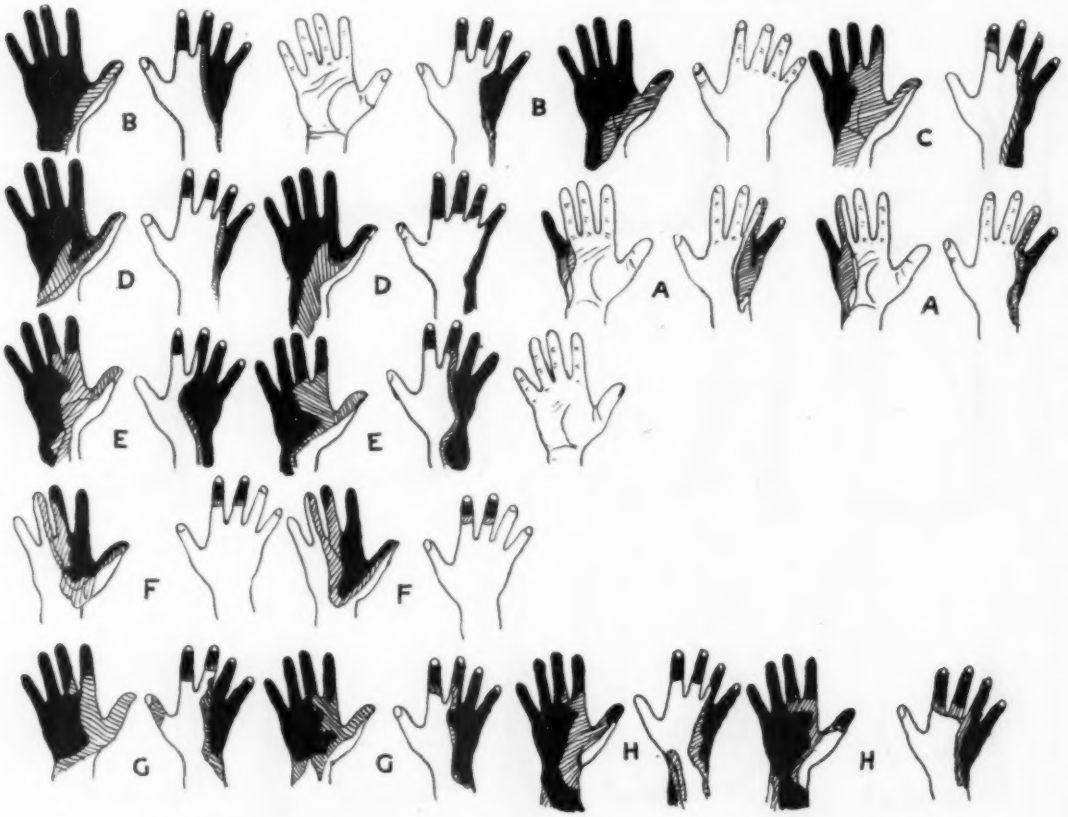

Fig. 13.—Sensory changes before and after resection and suture of the ulnar, median and ulnar and median nerves.

occupy the areas of nerve overlap, and that this return of sensibility to pin prick cannot be interpreted as a sign of nerve regeneration.

I am supported in this view by the facts that I have never found a return of sensibility to pain, when sensibility to touch has not returned, except in an area of overlap: that when a nerve is divided and at the same time one or more adjacent nerves are divided sensation to pin prick does not return in the area of the overlap of these

nerves even many months following the injury; that when a nerve adjacent to one which is severed and which supplies an area of overlap to that nerve is sectioned, the preexisting sensibility to pin prick in the overlap area is lost; that when sensibility to pin prick is present within the anatomic sensory distribution of a severed nerve resection and suture has no effect on the general outline of this area of sensibility.

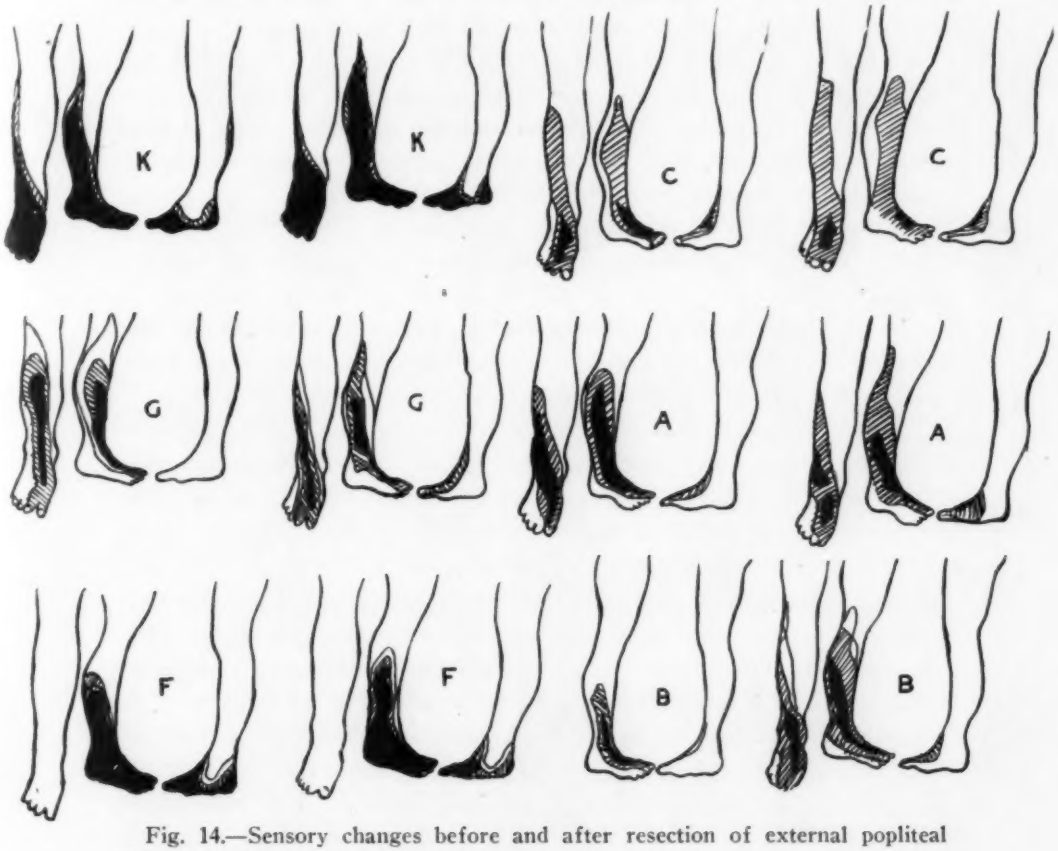

Fig. 14.—Sensory changes before and after resection of external popliteal and sciatic nerves.

The statement of Head, Rivers and Sherren[11] that "the length of the nerve to be regenerated makes relatively little difference to the time at which protopathic sensibility returns," only strengthens my thesis, for were the return of protopathic sensibility due to regeneration of protopathic fibers, this process of regeneration would have to conform with the same temporal law of regeneration as do the fibers carrying epicritic impulses, however rapidly the protopathic fibers might regenerate.

10. Head, Rivers and Sherren: Brain **28**:108, 1905.

Within two weeks after the occurrence of a peripheral nerve lesion the area of analgesia usually nearly coincides with the area of anesthesia. Some cases showed an intermediate zone or a shrinkage of the analgesic area within fifteen days. In from thirty to one hundred days the majority of cases showed the presence of a shrinkage to an extent which was later identified with overlap. It is probable that my cases would have shown the same extent of shrinkage in less than a hundred days, but conditions were such that in these cases the first record available was obtained one hundred days after the injury. Certainly the majority of cases showed the shrinkage to be well established under fifty days. Some months after the injury had been received the shrinkage was present and the remaining area of analgesia has been described above as the exclusive sensory supply for pain sense in various peripheral nerves.

The shrinkage of the analgesic area can be due to but two conditions — nerve regeneration or assumption of function by adjacent nerves.

If any overlapping of peripheral nerves is possible, it becomes necessary to define the extent of this overlap before any return of sensation can be interpreted as a sign of nerve regeneration.

Head, Rivers and Sherren[11] noted the presence of overlap of so-called protopathic sense, as the following quotation indicates: "Enormous overlapping occurs as we have already seen from a consideration of the analgesia caused by division of the median or of the ulnar nerves." Despite their recognition of overlap, they make no attempt to separate it from return of sensation due to nerve regeneration. Apparently they group both together when they say (page 108): "If the nerve has been completely divided, protopathic sensibility returns first, followed at a considerable later period by return of epicritic sensation." Speaking in this instance of recovery of sensation, they then go on to say without any reservation that: "Evidently, the two systems regenerate with unequal facility. The protopathic system regenerates more rapidly and with greater ease." Nor do Head and Sherren[12] clearly differentiate the two processes. Speaking of recovery of sensation after division of the nerves of the hand, they say, "But the ill defined borders and the comparatively small extent of total analgesia, and the fact that a large part of the palm rarely becomes insensitive to prick from a lesion of one nerve only, all point to much overlapping of the fibers that conduct pain impressions. Such overlapping should lead to rapid restoration of sensibility

11. Head, Rivers and Sherren: Brain **28**:110, 1905.
12. Head and Sherren: Brain **28**:148, 1905.

to prick, and in some cases possibly forms a factor when sensation returns with unusual rapidity. Commonly no wide loss to prick on the palm follows division of the median nerve, because the fibers which conduct this form of sensation are supplied from both nerves. But supposing the nerve supply of the median palm came overwhelmingly from the median, division of the nerve would produce at first total analgesia. This might rapidly pass away, to some extent, as soon

Fig. 15.—Sensory changes in lesions of median, internal cutaneous, combined median and radial nerve, *b, g, m,* from which the residual sensibility of the ulnar nerve was obtained; and of the ulnar and internal cutaneous, radial, combined radial and median nerves, *h, j, m,* from which the residual sensibility of the median nerve was obtained.

as the few fibers of the ulnar nerve to the median palm became capable of supplying sufficient sensibility for the transmission of impulses." Again they say (page 149): "With so much overlapping of nerve supply, complete recovery of sensibility to prick pain might occur without union of the divided nerve, by a further development of those

fibers in the uninjured nerve which normally supply the affected parts. In areas where sensation to prick is only partially lost, such substitution undoubtedly occurs, . . . But there is no evidence to show that restoration of sensation can be produced in analgesic parts without union of a divided nerve." Finally, they state (page 140) that, "Division of the nerve (one of the nerves of the hand) leads at once to the production of an area of absolute cutaneous insensibility, surrounded by an area of loss of sensation to stimuli, such as light touch and the minor degrees of temperature. The relative extent of these two areas differs greatly in each individual case, and the first definite sign of recovery is shown by an increase in size of the intermediate zone between them." So far as I can ascertain, no evidence has ever been adduced to show that overlapping nerves functionate immediately

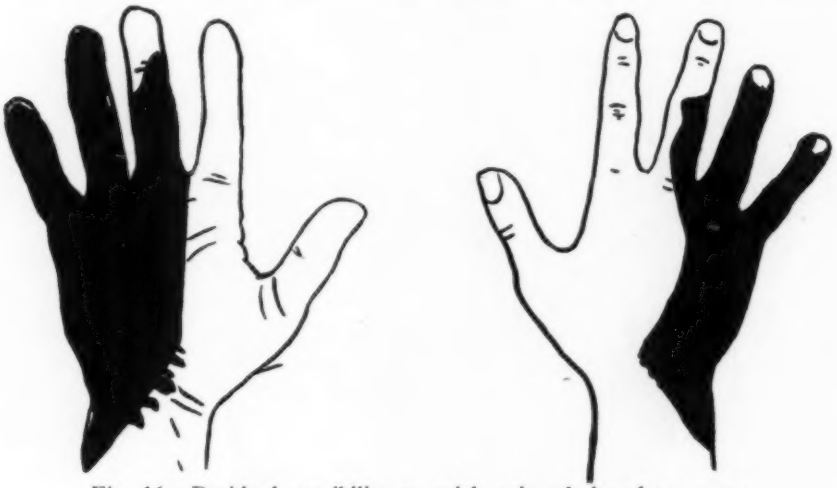

Fig. 16.—Residual sensibility to prick pain of the ulnar nerve.

following the injury of an adjacent nerve. Neither have the laws of dual innervation been clearly defined. Until this is accomplished, it is illogical to infer that return of sensation in the area of an overlapping nerve is a sign of nerve regeneration and is not caused by the functioning of this overlapping nerve.

If the shrinkage of the area insensitive to pin prick, responsible for the increase in size of the intermediate zone, be a sign of nerve regeneration and not a result of overlap, it should occur whether the adjacent nerves be intact or not. This, however, is not the case, as will be shown. In other words, if certain areas of skin become sensitive to pain or are found sensitive to pain following section of a given nerve, and the condition is due to nerve regeneration, then section of the adjacent nerve would have no effect on the appearance of this sensibility. What are the facts?

SECTION OF NERVES

Section of Adjacent Nerves.—Although in isolated lesions of the ulnar nerve sensibility to pain is frequently seen on the ulnar half of the ring finger, this is never observed when the median nerve is divided at the same time (Fig. 10, *a, f*).

Although isolated lesions of the ulnar and of the internal cutaneous nerves always show that the distal end of the analgesia resulting from a lesion of the internal cutaneous and the proximal end of the analgesia resulting from a lesion of the ulnar, do not meet, no instance is found in combined lesions of the ulnar, median and internal cutaneous nerves where an area between the borders of the analgesia of the internal cutaneous and ulnar nerves is sensitive to pain (Fig. 10, *b, c, d, e, h, i, j*).

Fig. 17.—Residual sensibility to prick pain of the median nerve.

When the ulnar, radial and median nerves are divided, a year may follow their division and no shrinkage of analgesia be found on the palmar or dorsal surface of the hand except on the proximal portion of the analgesia where the musculocutaneous and the antibrachii posterior areas adjoin the analgesic area (Fig. 11, *a, b, c*). When a radial lesion is combined with a median, analgesia is always present on the radial part of the palm. When a median lesion or a radial lesion alone is present, this part of the palm is usually sensitive to pin prick (Fig. 11, *d, e, f, g*).

Contrary to Head, Rivers and Sherren, who state that destruction of the external popliteal nerve (which corresponds closely to the fifth lumbar root) produces a widespread loss of sensation to prick, I have

found that isolated lesions of this nerve may show only a small area of analgesia, but when the internal popliteal as well as the external popliteal is severed, there is never found any shrinkage of analgesia or reappearance of sensibility to prick pain in the zone where the supply of the external popliteal meets that of the internal popliteal (Fig. 12, *a* to *g*).

I think it can be definitely stated that when nerves supplying adjoining areas are severed, sensation to pain is at no time present in the border areas where it is uniformly observed when either nerve is divided alone.

Inasmuch as a large number of my cases have had resections and sutures performed at least three months prior to the last examination,

Fig. 18.—Residual sensibility to prick pain of the musculospiral nerve.

it may be stated likewise that no sensation to pain returns in such areas in the time given for the beginning of regeneration of protopathic sensibilty.

Effect of Section of an Overlapping Nerve.—When return to sensibility to pain or presence of sensibility to pain is found in the area of overlap of an adjacent nerve, analgesia will result if this nerve is severed.

This is well illustrated in the case shown in Figure 3, *g*. This patient had a partial ulnar lesion combined with a complete section of the median. Prick pain was preserved in the radial portion of the palm and the index finger. When at operation the superficial radial

nerve was resected for use as a cable transplant, this part of the palm became analgesic (Fig. 11, *e*).

Effect of Resection and Suture on Existing Overlap.—Head and Sherren[13] state that "Sometimes it is necessary to divide an injured nerve, after sensibility to prick has already begun to return to the hand, that more perfect union may be obtained. Wherever such an operation has been performed, the parts that had begun to recover sensibility became again insensitive to prick, a proof that the recovery must have been due to union, however imperfect, of the divided nerves." This contention they support by Case 11 in which the ends of the divided nerve were united by what appeared to be fibrous tissue. After the operation, the extent of the total loss of sensibility to prick

Fig. 19.—Residual sensibility to prick pain of the musculocutaneous nerve.

had distinctly increased. If Figure 3, *b*, page 136, represents the sensory chart of this case after operation, a considerable area sensitive to pain within the area of anesthesia still requires explanation.

To have an increase of analgesia following resection and suture is not sufficent to prove the above contention. The whole area of anatomic sensory supply of a nerve must again become analgesic. That this did not occur even in Head and Sherren's cases is shown by Case 28, page 322. In a combined ulnar and median lesion the ends of the median nerve were found separated, but the ends of the ulnar

13. Head and Sherren: Brain **28**:108, 1905.

were connected by a thin strand of tissues. Following resection and suture, a considerable increase in the area insensitive to prick resulted. This increase occurred in the distribution of the ulnar nerve and may represent very well the change to a complete from an incomplete lesion. But even here the analgesia did not coincide with the anesthesia. The radial portion of the palm, the thenar eminence and the thumb remained sensitive to pain. This area, therefore, was the area of overlap of some other nerve to the median. Return of sensation in this area may be due to overlap, and never should have been considered in estimating the regeneration of this nerve.

My cases definitely show that, following resection and suture when sensibility to pain is present in an area of overlap, although some change in the outline of this area occurs, in general the area remains the same. At times the borders show some increase in analgesia; much more frequently they show a shrinkage of the analgesia. Slight changes in the borders of an area of analgesia cannot be used in arriving at a hard and fast conclusion. Frequently these borders change in an astonishing manner for pain produced by higher degrees of pressure by a sharp point not sufficient to produce pressure pain.

The laws governing dual innervation have not been clearly ascertained. What effect, if any, the handling of nerves or freshening of their ends may have on inhibition is unknown. In my opinion, another fact in support of the statement that return of sensation in an area of possible overlap cannot be ascribed to the regeneration of a nerve is that this area is not generally changed by resection and suture of a severed nerve.

The conditions necessary to study profitably the effect of resection and suture of nerves on return of sensibility to pain are: First, that the nerve ends be separated, and, second, that the examination subsequent to the operation be made within the period of time ascribed to the return of protopathic sense.

Some difficulty is encountered in meeting the second condition inasmuch as frequently the wide separation of the ends of the nerves makes it necessary to place the extremity in a position which will permit approximation, and to fix it in such a position by means of a case. This often prevents an examination before six weeks have elapsed. None of the cases here reported were examined later than fifty days after operation, one in less than fifteen days. Although some objection may be made to the cases examined over forty-five days after operation on the grounds of beginning return of protopathic sense due to regeneration, the similarity of the areas unaffected by operation in cases examined under forty-five days and those between

forty-five and fifty, coupled with the facts that the ends of the nerves were separated in all of these cases, makes it reasonable to admit them into the group.

To describe again the areas sensitive to pin prick in the lesions examined, or to attempt by description to show the sensory changes following operation, is needless. They are clearly indicated by

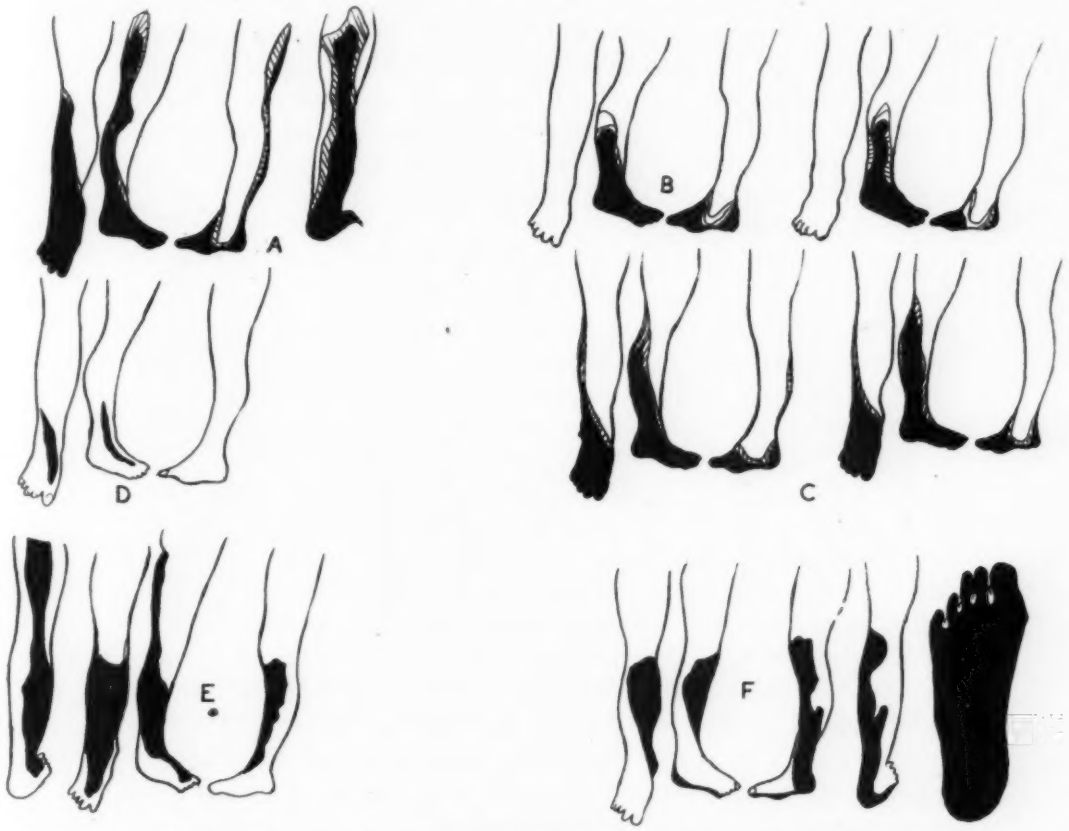

Fig. 20.—Sensory changes of combined lesions of internal saphenous and internal popliteal nerves, *f;* small sciatic, external popliteal and internal saphenous and sciatic nerve lesions from which the residual sensibility of the external and internal popliteal nerves was obtained.

Figures 13 and 14. It is sufficient to state that the following nerves were studied: ulnar, examined forty-two days after operation, Figure 13 *a;* median, eight days after operation, Figure 13 *f;* ulnar and median, forty-five, thirty-six, forty, forty-six, forty-eight and fourteen days after operation (Fig. 13 *b, c, d, e, g, h*); external popliteal, forty-eight, thirty-six, twenty and twenty-six days after operation, Figure 14 *a, b, c, d;* sciatic, fifty and thirty-six days after operation, Figure 14 *f. h.*

3. RESIDUAL SENSIBILITY

If we assume the relatively early return of sensibility to pin prick to be due to overlap it becomes possible by the method of residual sensibility to outline the borders of overlap of the various peripheral nerves.

The method of residual sensibility is based on the assumption that following section of a given nerve, the area of skin, in its anatomic distribution in which sensation remains, is subserved by the intact adjoining nerves distributed to that area. For example, four nerves supply the palmar surface of the hand: the ulnar, median, musculocutaneous and radial. If two — the ulnar and median — are severed, what sensibility remains is subserved by the musculocutaneous and radial. If then the borders of the musculocutaneous be determined, that which remains is radial.

In employing this method certain precautions must be observed. For example, we cannot take the outer border of the analgesia on the dorsal surface of the hand in an ulnar section to be any part of the border of the overlap of the median unless we may observe the effect of a combined ulnar and musculospiral so that the overlap of the latter nerve be not included. Similarly, we cannot outline the border of the overlap of the median on to the radial unless we have had a combined lesion of the ulnar and radial to indicate the distribution of the ulnar; or the overlap of the radial to the median on the palm unless we have had a combined median and musculocutaneous lesion, or the overlap of the internal popliteal to the external popliteal unless we have had a combined lesion of the external popliteal with the internal saphenous. The necessity for these combinations reduces the number of cases available for conclusions to a very few. As a result the areas of overlap as outlined probably were smaller than the real overlap. However, the extent of overlap was sufficiently large to prove my contention that it is within such an area that return of sensibility to pin prick occurs soon after injury of peripheral nerves.

Owing to this clinical lack of certain combination injuries, I have been unable to determine all the overlap areas of some of the nerves investigated.

It is hardly necessary to state that the cases studied must have nerves recently resected or be examined prior to an operation which reveals the ends of the nerves separated.

In illustrating the areas of overlap, the space between the borders of the overlapping to adjacent nerves has been blocked out with black. The black area, therefore, represents the total supply to pain of the various nerves studied. The area of actual overlap would be that part of the total sensory supply to pain which extends beyond the

accepted sensory limit of the adjacent nerves. The restrictions of the methods necessary to obtain these areas are responsible for an indicated area of total supply, which in some instances is smaller than is actually present, as may be seen in the case of the outer border of the ulnar on the dorsal surface of the hand and the inner border of the external popliteal on the back of the leg (Figs. 17 and 21).

The area of total supply to pain of the ulnar nerve was obtained by the method of residual sensibility from a median lesion, an internal cutaneous lesion and a combined median and radial (Fig. 15, *b, g, m*). It occupies the ulnar portion of the palm to a line which is a continuation of the ulnar border of the abducted index finger, the palmar surface of the fingers except the terminal phalanx and one-third of the ulnar part of the second phalanx of the middle finger. On the

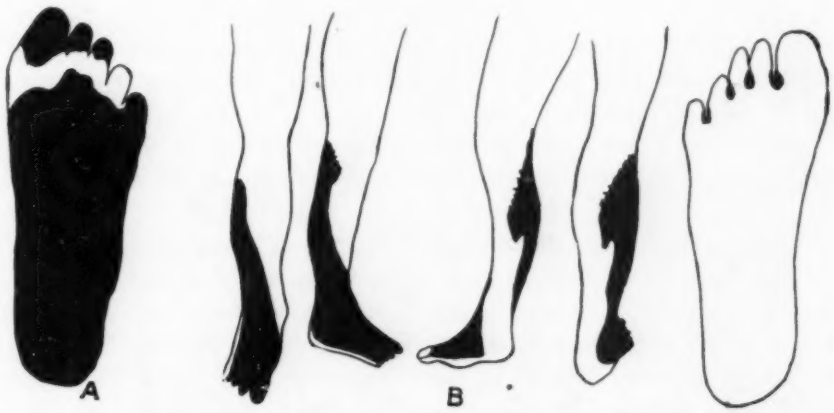

Fig. 21.—Residual sensibility to prick pain of external popliteal nerve, *b;* sensory changes in an uncertified case of complete interruption of the internal popliteal, *a.*

dorsal surface it occupies the ring, little and more than the ulnar half of the proximal one-and-a-half phalanges of the middle finger, the dorsum of the hand to the radial border of the fourth metacarpal bone, ending proximally one inch above the wrist (Fig. 16).

The area of the median nerve was obtained from an ulnar and internal cutaneous lesion, a radial, a combined radial and median lesion and cases of combined radial and ulnar lesions (Fig. 15, *h, j, m*). The inner border on the palmar surface was obtained by the method of residual sensibility from an ulnar and internal cutaneous lesion. On the dorsal surface I was compelled to employ another method, as the cases of combined ulnar and radial lesions were too recent to have had return of prick pain due to overlap. The border of overlap of the musculocutaneous to the radial was obtained by means of residual

sensiblity in a case of combined radial and median. Inasmuch as the radial has no isolated supply to prick pain, this border separates the musculocutaneous from the median overlap. I, therefore, used this border as the proximal border of the median overlap to the radial nerve. We were supported in doing this in such cases as showed an area of analgesia between the areas of overlap of the median and musculocutaneous nerves (Fig. 15). Part of the inner border of the overlap on the dorsum of the hand is hypothetical and shown as a rough border (Fig. 17).

The total supply of the musculospiral nerve was obtained from cases of combined ulnar and median lesions, a case of combined ulnar, median and musculocutaneous lesions and a case of combined ulnar an internal cutaneous lesions (Fig. 15, *a, d, e, f, h, i, k, l, m, n*). I found an overlap on to the palm to an extent heretofore undescribed. In median nerve lesions the sensibility to pain in the palm has frequently been ascribed to ulnar overlap. But Athanassio Benisty[9] recognized the importance of the musculospiral and the musculocutaneous nerves in this condition.

The area of overlap on the palm of the musculospiral nerve extends over the radial part of one-and-a-half phalanges of the index finger, the radial part of the proximal phalanx of the middle finger, and the web between the middle and ring fingers, all of that part of the hand external to a line continuous with the radial border of the middle finger. Internally, it extends from the middle of this line to the middle of the base of the first phalanx of the ring finger and proximally to the middle of the outer surface of the wrist, from which point the border extends in a line to a point one inch proximal to the base of the metacarpal bone of the thumb on the radial border of the wrist. This area occupies the entire dorsal surface of the hand, with the exception of a strip one half the width of the little finger on the ulnar border, the little finger, the distal two phalanges of the ring finger, most of the distal two phalanges of the middle finger and a little more than the distal phalanx of the index finger. The area on the forearm need not be described (Fig. 18).

The inner border of the pain area of the musculocutaneous nerve on the anterior surface of the forearm was obtained from the residual sensibility following section of the internal cutaneous nerve; the inner border on the dorsal surface of the forearm, from a musculospiral division. The distal border on the palm was obtained from radial lesions, a combined radial and median lesion, and from lines obtained in combined ulnar and median lesions where an area of analgesia existed between the areas of overlap of the musculospiral and the musculocutaneous (Fig. 15, *e, f, k*). On the dorsal surface of the

hand combined sections of ulnar, radial and median and a case of combined radial and median were employed (Fig. 11, *a, b, c, f;* Fig. 15, *m, h, j*). The area of total sensory supply to pain of this nerve can be better appreciated by viewing the illustration than by description (Fig. 19). The proximal limits of both the musculocutaneous and musculospiral nerves are hypothetical.

I was fortunate in obtaining two cases from which the overlap of the internal and external popliteal nerves could be obtained according to the method of residual sensation. One was the case in which the internal saphenous and internal popliteal nerves were injected with alcohol for causalgia, producing anesthesia, the residual sensibility about which permitted the outlining of the total supply for pain of the

Fig. 22.—Residual sensibility to prick pain of the internal popliteal nerve.

external popliteal nerve (Fig. 20, *f*). The upper border of this area is hypothetical and merges on the outer surface with the external cutaneous, on the posterior surface with the small sciatic and on the inner side with the obturator nerve.

The overlap on the sole seen in Figure 21 *b* is probably smaller than that which actually exists, as may be seen from the presence of sensibility to pain in the blank area of the sole in a case of complete interruption of the internal popliteal nerve, a case which, because it was not certified by operation, is not included in my series (Fig. 21, *a*).

The area of total supply for pain of the internal popliteal nerve was obtained from the residual sensibility in a case of a combined lesion of the small sciatic, the internal saphenous and the external

popliteal nerves, and a case of external popliteal section (Fig. 20, *d*, *e*). The upper border of this area is hypothetical and merges with the borders of the small sciatic and obturator nerves (Fig. 22).

The area of total pain supply of the internal saphenous nerve was obtained from the residual sensibility of a combined lesion of the small and great sciatic and cases of sciatic section (Fig. 20, *a*, *b*, *c*). The upper border here is likewise hypothetical, merging with the borders of the anterior crural, the external cutaneous and the obturator nerves (Fig. 23).

THEORETICAL CONSIDERATIONS

The evidence adduced above to show that the early return to sensibility to pain cannot be interpreted as a sign of nerve regeneration, but probably occurs as the result of nerve overlap, does not invalidate that part of the theory of Head and his co-workers which relates to the existence of a distinct system of fibers for epicritic and for protopathic sensibility. The question of the existence of these two systems can probably be settled only by psycho-physical research.

Boring[7] defines in part the sensory changes following section of a sensory nerve as follows: "We have seen that in the outer zone of the affected region there is at any sensory spot (the region stimulated by a punctiform stimulus) a partial loss of sensitivity. Since the partial loss comes as the result of the complete severance of certain fibers, we may presume that the partial function remaining occurs by way of other fibers which have not been cut (principle of multiple innervation of a sensory spot). These must come from an adjacent nerve."

I do not believe that any hypothesis of dual innervation is immutable. The probability that the return of so-called protopathic sense before the return of epicritic sense is due to nerve overlap and not to a temporal dissociation of protopathic and epicritic sensibilities attacks one part of the hypothesis of Head and his co-workers. This emphasizes the necessity for review of the theories of dual innervation and for that reason I shall quote freely from Boring (p. 89):

> Suppose, now, that we have a spot which is innervated primarily from the nerve *P*, but which is also innervated secondarily by fibers from the nerve *S*. Let such a spot be *X*, Figure 97, *a*. Since *P* and *S* are from adjacent nerves, they will be projected upon adjacent regions at the center; and, since *P* is primary and *S* secondary, the corresponding excitations, *p* and *s*, will be unequal — that is to say, *p* will be greater than *s* (Fig. 97, *a*). Thus *s* may be subliminal and *p* supraliminal, so that *p* alone is adequate to sensation. We may assume this to be the normal state of affairs; namely, that the primary fibers are alone effective, and that their effect is always partially decreased by the secondary effect (for the secondary fibers, projected upon a different region, would tend to inhibit the primary: . . .). .

Now let the nerve P be cut (Fig. 97, b). At once the inhibiting effect of p upon s is removed and s is increased. It may, however, still remain subliminal. Thus it appears that, even in an area of complete sensory loss, there may be fibers from adjacent nerves which tend to function, but which are not adequate to a supraliminal excitation.

As time goes on, the secondary fibers would tend to take on the function of the primary fibers (principle of vicarious function). The many cases of gradual recovery from permanent lesion have shown that, whenever possible, an interrupted function is taken over by other elements which are spatially or functionally connected, and that the vicarious assumption is not immediate but gradual—by practice. Thus, as the S—fibers became more "practiced," the s—excitation would become first supraliminal (Fig. 97, c), and then, since there is no p— excitation to inhibit it, it might become more intense than the p— excitation was originally (Fig. 97, d).

Fig. 23.—Residual sensibility to prick pain of internal saphenous nerve.

The next stage would occur when the P— fibers began again to make functional connection (Fig. 97 e). The s— excitation would be diminished by the small, subliminal p— excitation. The return of functional adequacy to P would doubtless be gradual, but eventually p would become effective for sensation, and two sensations (Fig. 97, f) would be felt. If now P is so situated as to form better or more connections with the sensory spot, x, than is S, then it will come to dominate S in its demand upon central energy; and S, now less effective, will undergo the functional decay, which is the converse of "practice" in vicarious functioning. Thus, from the conditions of Figure 97, f, the original normal conditions of Figure 97, a, would finally be recovered.

The significance of the figures on the introspective side requires only a word. At first, stimulation of x gives rise to a single sensation, normally localized (Fig. 97, a). Then the nerve is cut and there is no sensation (Fig. 97, b). Then stimulation of x produces a weak sensation, remotely referred with regard to the localization of the original sensation (Fig. 97, c). The sensation becomes abnormally intense (Fig. 97, d); then decreases (Fig. 97, e); and then

is supplemented by another sensation referred to the original point (Fig. 97, *f*). Finally, the normal condition recurs (Fig. 97, *a*). Such would be a course of returning sensibility, although it is not necessarily the typical course.

On our hypothesis it is not possible that all the fibers of any one nerve are projected upon the same central area. In dividing a nerve, we must divide both primary and secondary fibers for many sensory spots. Such a condition is diagrammed in Figure 98. The normal excitation (Fig. 98, *a;* compare with Fig. 97, *a*) is entirely abolished by the section of both the fibers *P* and *S* (Fig. 98, *b*). Since the primary fibers are probably in a better position to make functional connection than are the secondary, they would be the first to become effectively connected; and we should have the subsequent stages of Figures 98, *c* and 98, *d*. If the secondary fibers did not return rapidly to function, the *p*— excitation, relieved of their inhibiting effect, might rise above normal (Fig.

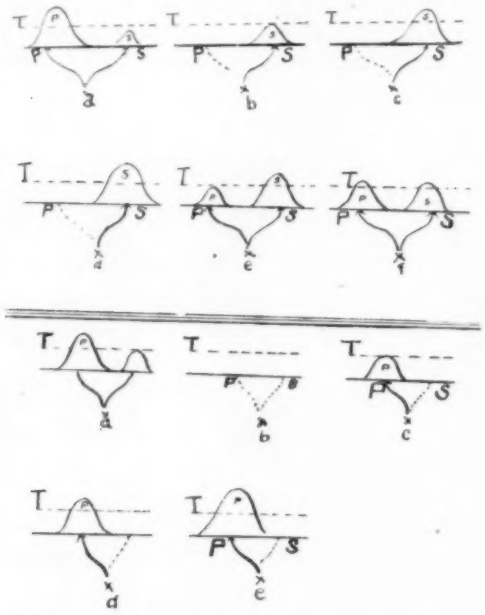

Fig. 24 (Figs. 97 and 98, Boring).—Successive changes with respect to intensity and reference of sensation, dependent on the stimulation of a single point, dually innervated after the division of one (Fig. 97) or both (Fig. 98) of the innervating fibers (see text). Compare Figures 94-96. Dually innervated point on the skin, *x;* primary innervating fiber, *P;* secondary innervating fiber, *S;* corresponding central excitations, *p, s;* central threshold, *T*.

98, *c*). The subsequent return of the *S*— fibers would, however, eventually bring about the normal state again (Fig. 98, *a*). Here, then, we have a case in which recovering sensibility passes through a stage of abnormal intensity, without, however, being abnormally localized.

Results of my investigation permit criticism of only that part of the theory of Head and his co-workers dealing with the temporal dissociation of epicritic and protopathic sensibility.

CONCLUSIONS

1. The area of prick pain supplied exclusively by an individual nerve is far less than the accepted sensory distribution of that nerve.

2. The area between the border of exclusive supply of prick pain of an individual nerve and the border of its accepted sensory supply constitutes the area of algesic nerve overlap.

3. When nerves serving adjacent areas are severed, sensibility to prick pain between these areas is not present after injury, nor does it return before the sense of touch.

4. When a region in the area of sensory distribution of a severed peripheral nerve is sensitive to prick pain, and this region is adjacent to another nerve area, if this second nerve be severed, complete analgesia results in the previous sensitive region.

5. When sensibility to prick pain is present or returns in the area of possible overlap on to the sensory distribution of a severed nerve, subsequent resection and suture of this nerve does not change the general extent of this sensitive area, although the borders may at times be slightly enlarged or diminished. That is, the pain sense returned or present before the operation was not due to partial regeneration.

6. The laws governing the assumption of function by nerves adjacent to a severed nerve are unknown.

7. Handling and resection and suture of previously divided nerves changes the condition governing the function of overlapping nerves, often initiating greater function.

8. Evidence of the assumption of function by nerves adjacent to a severed nerve is not present immediately following the nerve injury, but gradually shows itself at a later date.

9. The early return of sense of prick pain before the return of sense of touch is not due to temporal dissociation of epicritic and protopathic sensibilities, but is due to the assumption of function by adjacent overlapping nerves.

10. The areas of overlap may be determined with fair accuracy and the early return of sense of prick pain in those areas cannot be interpreted as a sign of regeneration of the divided nerve.

11. The changes in prick pain following division of a single nerve are not a safe basis for conclusions regarding regeneration of that nerve.

12. Only when a group of nerves is divided at the same time can the studies of sensation be used in the interpretation of regeneration of these nerves. Under these conditions only that part of the analgesic

area may profitably be studied which is removed from the effect of overlap from adjacent nerves. On the other hand, if return to sensibility to prick pain occurs on the border of an uninjured adjacent nervé, this return to sensibility does not indicate regeneration of a nerve.

13. Return of sensibility to prick pain can be used clinically for the determination of nerve regeneration only when it is accompanied by return of tactile sense or when it occurs outside the area of possible overlap of adjacent nerves.

In conclusion, I wish to express my thanks to Lieut.-Col. Dean D. Lewis, whose encouragement and interest made this work possible, and my gratitude to Capt. Wesley Gatewood, who, by his industry and enthusiasm, contributed no small part of the matter contained in this communication.

Abstracts from Current Literature

ASCESSI E TUMORI DELL'ENCEFALO. Osservazioni Cliniche Ed, Anatomopatologiche del dott. G. MINGAZZINI, Ordinario di Neuropatologia nell Regia Universitá di Roma, Riv. di patol nerv. **24**: Parts 3-4 and 5-8, 1919.

The author has written an extensive article, which appears in two numbers, and merits this rather long review. He mentions the numerous diagnostic difficulties, particularly as to localization of brain abscesses and tumors, and ascribes the poor surgical results which surgeons obtain in these cases to the facts that first, the neurologist is unable to give always accurate localization of the process and second, the diagnosis is not made early enough. In brain abscesses, even when evacuation is possible, death will occur because of the edema which involves vital areas or because of the "marasmus" which has begun. We do not possess unfailing guides to differentiate cortical from subcortical tumors. Unfortunately, exactly this differential point is necessary beforè the operator can use surgery with success. It is not enough to tell a surgeon that the tumor is frontal or temporal, for these areas are too extensive to allow complete exposure at operation. These cases must be minutely studied and more accurate localization arrived at. In tumors and abscesses of the cerebellum, however, this is as yet impossible and very wide exposures are advised by Duret and Murri. By this method one is able to explore for tumors with the finger, but it is only by means of exploratory puncture in various directions that one can locate the site of abscesses in the cerebellum. Murri states that the number, progression and character of the symptoms produced by tumors of the frontal and temporal lobes are so varied that no one can lay down fixed rules. Nevertheless, the author believes that constant attempts should be made to study the symptoms, and thus definitely localize the process.

The author's material consisted of four abscesses of the cerebellum and eight brain tumors, three of which were in the posterior fossa, three in the middle and two in the frontocentral fossa. In localizing a cerebellar abscess, the difficulties are increased because of the almost invariable presence of mental confusion which interferes with the delicate tests necessitating good cooperation or because, as often happens, an abscess may occur in a patient who has double otitis media. With these preliminary remarks, the author describes in detail his four cases of cerebellar absceess. In order to know exactly how he observes his material, the first case is reported in some detail.

The patient had an abscess in the right cerebellar hemisphere, was operated on and died. Syphilis was denied. There was a history of acute otitis media, with a cessation of discharge from the right ear; a few days later he had headache (occipital), more severe on the right side and when standing. The patient became rapidly semistuporous, but on being transferred from the ear department to the author's service, he brightened up enough to give his history. The temperature had always been subnormal. On Feb. 28, 1918, four weeks after the onset with headache, there was rigidity of the neck, especially on movement to the right; pressure on both jugulars did not cause pain; there was limitation of mobility of the right eye to the right; there was no interference with other movements. There was a slow nystagmus, especially on

looking to the left, a mild right facial weakness, but no limitation of tongue movements or deviations. The patient was not able to hold his hands upright in the attitude of a person who is being sworn, both falling rapidly; the right extremity oscillated considerably as it fell. This attitude is one which the author mentions as a test for paresis in the upper extremities, and he apparently thinks it is an important test. There was diminished resistance to passive movements on the right side, and both lower limbs could not be held raised in the air. There was decided weakness, therefore, in both the upper and lower right extremities, but active movements were well made. Both pupils were widely dilated, and light reaction was absent. The tendon reflexes on the right side were much diminished, but were quite active on the left. There was plantar flexion in both big toes; abdominals were active, and the right corneal was diminished. Percussion of the skull was painful in the right occipital region; pain, temperaure and tactile sensibility were unchanged; the finger to nose test on the right side was badly done, but asynergia could not be tested for, on account of the patient's mental condition. The patient could stand alone only on a wide base with his feet widely apart. He could walk if supported, swaying to the right or left; if not held, he fell, usually toward the left. He lay on the right side, in bed, with head bent backward; when he arose the headache was worse; he past pointed spontaneously to the right with the right finger; sense of taste and smell were not tested. There was a bitemporal pallor of both disks; pulse, 60; urine, negative. The patient cried, shrieked and was semi-stupid. He was oriented for time, place and person. Attention was poor and perception not good. Lumbar puncture gave a few drops of turbid, bloody fluid under slight pressure. No fluid could be aspirated with a syringe.

The patient died Feb. 28, 1918, and a necropsy was immediately done. The right cerebellar hemisphere was found much swollen, the lobus quadratus soft in consistency and only when frontal sections were made, did a fetid pus escape from the ventral surface of the hemisphere, leaving a cavity which took in the subcortical area corresponding to the lobus quadratus and lobus semilunar posterior. There were two other necropsies, making a total of three of four cases of cerebellar abscesses and in the fourth, the diagnosis was verified at operation.

DISCUSSION

Oppenheim and Cassirer are referred to by the author as authorities for the statement that abscesses in the posterior fossa cause the most intense occipital and nuchal pain, but occasionally frontal and rarely parietal and temporal headaches occur. In the author's Case 4 (abscess of the anterolateral left hemisphere of the cerebellum) headache was in the left frontal and temporoparietal region. In his Cases 2 and 3 headache was not localizable but constant (abscesses of antero-medial and antero-inferior portions of cerebellar hemisphere). In Case 1, headache was worse on the right side and in erect posture (abscess of dorsolateral portion of the right cerebellar hemisphere). From the author's cases, it would appear that headache is diffuse if the abscess is central and inferior, but is worse in occipital region on the same side as location of abscess. Mingazzini warns against using pain as a localizing sign without careful consideration of other symptoms. Oppenheim and others assert that tenderness to percussion and pressure is worse when applied to the same side as the lesion and usually corresponds to the site of maximum headache. This was true in the author's cases. Mingazzini also confirms Oppenheim's finding of dizziness and vomiting as early symptoms, these occur-

ring in all four cases, most marked, however, in Cases 1 and 2. Oppenheim finds that optic neuritis (blurring hyperemia, loss of outlines) occurs in cerebellar abscess frequently; not infrequently a papilledema may occur, always worse on the side of the abscess. The author says, however, that a number of cases occur which never show fundus changes at any time in their course (his Cases 2 and 4 are cited). In the other two cases there was bitemporal pallor in one, optic neuritis in the left eye and papilledema in the right eye (same side as abscess) in the other case. Acuity of vision was retained in all four cases, but blindness may occur. All his cases showed the following mental signs: clouding of the sensorium, poor perception and attention and in one case disorientation for time. In Case 1, there was early stupor. Only in episodes does one see restlessness and excitement. The pulse is usually slow.

None of his cases had cerebellar seizures (attacks of vertigo, nystagmus, very severe headache and paresis). The author's cases confirm the statement that in cerebellar abscess, nystagmus is usually toward the side of the abscess. In Case 4, the movement was slow, rotary and appeared only when the patient looked toward the affected side. In Case 1, however, this appeared only when the patient looked away from the side with the abscess. There was paralysis of gaze or of one sixth nerve alone, usually homolateral. In Case 2, the sixth pair was affected, probably because in this case the pus tended to collect in the base in the median line. In Cases 3 and 4, there was a paralysis of the twelfth cranial nerve, the tongue deviated toward the side of the abscess in one and away from it in the other. The author states that it is difficult to say whether this paralysis is due to compression of the nucleus of the twelfth nerve in the medulla or of the supranuclear pathways, but thinks it is probably due to both; that is why the tongue deviates sometimes toward and sometimes away from the lesion. The seventh pair of nerves in the author's cases were paralyzed on the same side as the abscess, the explanation being that pressure is exerted by the abscess at the base as the nerve courses there. The author does not believe that suppurative processes in the internal ear cause the seventh nerve palsies. Ataxia and adiadokocinesis was present on the same side as the lesion in two of his cases; but in Case 1, when the patient walked, there was a tendency to fall to the side opposite to the lesion. Many authors state that this symptom is always toward the side of the lesion, and this is usually true; but in this case the vermis was undoubtedly involved and therefore the cerebellospinal pathways were interefered with, causing the swaying toward the opposite side, but in Case 2, the underlying pyramidal tract was injured causing the tendency to fall toward the side of the lesion. In only two cases was ataxia present, but in explaining this, the author points out that weakness often interferes with tests for coordination; also that the mental condition interferes with the sensory examination. Nevertheless, he was able to find hypesthesia in the distribution of the right fifth nerve in Case 2, and in Case 4, hypesthesia including pressure and vibratory sensibility and some degree of astereognosis on the same side as the abscess. He never found hyperesthesia. In Case 4, the abscess undoubtedly pressed on the tegmentum pontis because of its situation in the left anterolateral hemisphere of the cerebellum. Only in one case (2) was there any Babinski phenomena (fanning). In Case 3 (abscess on antero-inferior surface), the patellar and tendo-achillis reflexes were entirely abolished on both sides and in Case 2, much diminished on the side of the abscess. Whether this rule of diminished reflexes holds good, depends entirely on the degree of involvement of the pyramidal tracts. The corneal reflex was diminished in only two cases, in

both occurring on the same side as the abscess. The size of the pupils follows no definite rule, and is therefore not helpful in localizing. The mydriasis or myosis depends on the degree of pressure endured by the third cranial pair of nerves; double ptosis was present in one case. Exophthalmus, depending on hydrocephalis, occurred on the same side as the abscess in one case. Dysarthria from pressure on the pons and medulla is rarely seen, but was present in Case 4. Attempts to aid in localization by attention to errors of pointing and past pointing should be discounted by the difficulties of cooperation because of the patients' mental condition, weakness and possible loss of position sense. In two of the author's cases, deviations in past pointing were never constant. In Case 1, the patient past pointed to the right; the lesion was in the right dorsolateral hemisphere. This does not seem to bear out Bárány's claim, that past pointing to the right in the right upper extremity is localized in the inferior and superior semilunar lobes of the cerebellum. They were intact in this case.

All four patients died. Three were operated on, the pus was evacuated in two, and not found at operation in one. The delay was fatal in all cases. In Case 4, the abscess drained well and improvement occurred, but death followed due to the formation of another abscess. According to Oppenheim and Cassirer, this often happens—the abscess is evacuated but later another develops. The author advises puncturing the cerebellum in many directions until pus is found. In one of his cases, at first operation punctures failed to find pus; a few days later, the needle being introduced in other directions, much pus was found.

The most constant signs of cerebellar abscesses are: mental clouding, headache, rigidity of the neck, vertigo, vomiting and changes in the fundi. Less frequently there is cerebellar ataxia, fever and marasmus. The localizing signs are usually found on the same side as the abscess: pain on percussion, hemi-ataxia which is static or dynamic, adiadokocinesis, hemihypesthesia, areflexia of cornea; less frequently, myosis, sluggish reaction of pupils to light, optic neuritis, error of past pointing and change in the deep tendon reflexes. The latter, however, together with deviations of the tongue and the position in which the patient lies, when in bed, cannot be used as aids to localization.

The author then presents his cases of tumor of the brain. The first one—Case 5—is a case of tumor of the right cerebellar hemisphere, tegmentum pontis, et bulbi. This case was particularly difficult because for a long time there was a justified suspicion that it might be a case of cerebellar abscess. The boy, 11 years of age, had had chronic otitis media for several years until May, 1917, when the discharge from the right ear suddenly stopped; then began headache, vomiting, vertigo (objects whirled from left to right). The headache was most severe at the vertex and left parietal region. There were slight elevations of temperature (37.2) daily, with chills. In June the patient had diplopia, gradual loss of vision and loss of hearing in the right ear, together with disturbances of gait. The pulse was 66, lumbar puncture showed a fluid under great pressure, negative "sediment and Nonne, 1 per cent. albumin and no coagulation." The findings were: headache, vomiting, dizziness, fever, mental dulling, papilledema, exophthalmos in the right eye, paralysis of all the cranial nerves, including the fifth to the twelfth, hypesthesia and hypothermalgesia in the faciobrachial distribution with loss of vibration sense in four toes of the right foot, hypotonia and static ataxia of all the extremities, restriction of the right visual field, diminution of the

sense of smell, diminished reaction of both pupils to light, absent patellars and tendo-achillis reflexes diminished, areflexia of the cornea and spontaneous past pointing to the right. The patient had a tendency to lie on his left side. Cranial percussion was more painful in the left parietal occipital region; there was bilateral nystagmus and cerebellar gait with a tendency to fall toward the left.

At necropsy a white swelling was found in the middle of the gray matter of the right cerebellar hemisphere which was continued downward into the right half of the pons where it was almost impossible to make out the formation of the tegmentum, particularly of the lemniscus and the formatio reticularis. In discussing this case, the author mentions the fact that the location of the process in the cerebellum, pons, and medulla of the right side was suspected from the beginning, despite the fact that there was no paralysis of gaze. But he agrees with Murri that this symptom is not constant in tumors of the pons because alternating palsies of the eye muscles occur which are sufficient in themselves for intrapontile localization. Cerebellar abscess was suspected chiefly on account of the sudden cessation of the discharge from the right ear and the slight elevation of temperature, with great loss in weight. The author points out that while hypotonia existed in the right extremity, the patient in walking fell toward the left; this he explains by the displacement of the vermis obliquely toward the right, injuring in this way the pathways proceeding from the right cerebellar hemisphere and thus interfering with the tone and taxis of the right side. In order to explain the right faciobrachial sensory changes, an invasion of the left side of the tegmentum pontis must be supposed, involving a great part of the trigeminus and the lemniscus on that side. Particular mention is made of the fact that the patient preferred to lie on the left side, and suffered most pain in the left parieto-occipital region on the side opposite the tumor. While this is not usual, Bruns explains it by pointing out that cranial percussion is most painful in tumors of the cerebellum associated with marked hydrocephalus, while diffuse pain is most common with tumors in the cerebellar hemispheres near or on the cortex. The author explains the presence of a pain on the side opposite the lesion by ascribing it to the compression exerted on the healthy hemisphere and its dura by the diseased side.

Concerning his sixth case, which was a tumor of the tegmentum pontis on the left side, the author says that the progression of the symptoms suggested at first a neoplasm of the cerebrum even though there were no disturbances of the fundi or of vision. The dominant symptoms at first were: paralysis of gaze toward the left, weakness of the left internal rectus and of the seventh nerve, and on the right, an alternating paralysis of the extremities with hypesthesia of both superficial and deep sensations. The latter symptoms made it probable that the tumor was situated in the anterior portion of the left tegmentum pontis, and that it extended forward toward the pyramid. The absence of the left corneal reflex and the spontaneous error of past pointing of the right hand to the right made one suspect that the tumor had begun in the left posterior fossa and then had compressed the right cerebellum. Later, however, when adiadokocinesis, ataxia, cerebellar gait, etc., had made their appearance, it was suspected that not only the tegmentum, but also the brachium pontis of the left side had been invaded. The author believes that the various forms of sensibility run through the tegmentum pontis in separate groups—the stereognostic sense fibers through the middle of the lemniscus, tactile sensibility through the posterior zone of the lemniscus close to the

formatio reticularis, while the pain fibers and those of heat and cold seem to run through the formatio.

In his seventh case, which was a tumor of the left cerebellopontile angle, the author discusses the characteristic triad of this syndrome—disturbance of function of the fifth and eighth nerves with nystagmus. In the author's case there was also involvement of the third and fourth nerves. In tumors of the pons itself, the eighth nerve is less often involved and usually much later than in tumors at the angle. Also, papilledema is less frequent and the course of the disease more rapid. Some difficulties in the diagnosis of the author's case were found on account of the presence of weakness and atonia on the right side opposite to the pareses of the cranial nerves, such as often happens in pontile tumors, but the unilateral character and the early appearance of cochlear and vestibular symptoms, together with the more marked papilledema on the left side, localized the tumor. The exophthalmos was also more prominent on this side. Concerning the cochlear and vestibular functions, Mingazzini refers to many authorities who agree with him that while the cochlear functions are never destroyed so that complete loss of hearing occurs, yet vestibular excitability of the affected side may be completely lost and diminished on the sound side. Three cases are referred to in which the vestibular functions were excitable also on the side of the tumor. The author believes that a diagnosis of cerebellopontile angle tumor can be made whenever there are disturbances of function of the fifth and eighth nerves, paralysis of the sixth and seventh, nystagmus, paralysis of lateral gaze, exophthalmos, hypogeusia and the tendency of the body to fall toward the suspected side, with increase of cranial percussion pain on the affected side. Location of weakness, atonia and disturbance of reflexes are considered of little importance because in some cases the dentate nucleus is irritated, while in others it is destroyed, which accounts for the difference in symptoms. In tumors of the cerebellum, however, the side on which disturbances of the cranial nerves are found is not necessarily the side of the lesion because the tumor may be exerting its pressure in an oblique contralateral manner, compressing the other side. Particular attention must be paid to the progression and history of the onset of the various symptoms. The author agrees with Cushing in laying great stress on paralysis of the peripheral type of the seventh nerve as an important localizing symptom of tumors of the angle. This may exist alone for a long time before other symptoms occur. It is possible that only one branch of the seventh nerve may be involved. The author lays great stress on this point.

In these three cases (5 to 7) the author points out the paucity of mental disturbance in tumors of the posterior fossa as contrasted with the more marked mental disturbance in tumors of other parts of the brain. In his case of angle tumor the author also directs attention to the fact that there was great variation in the spinal fluid findings, not only as to pressure, but some cases have also been described in which the Wassermann and globulin reaction was positive. He says that a positive Wassermann is frequently found in nonsyphilitic tumors of this region.

Case 8 was a tumor of the median portion of the fovea cerebralis media dextra. The patient began with a progressively increasing headache, dimness of vision, vomiting and hiccup. The roentgen ray disclosed disintegration of the sella. On the right side there was almost complete ophthalmoplegia, hypesthesia of the face, mydriasis, painful percussion and blindness. On the left side, there was weakness of the extremities, increase of the deep

tendon reflexes of the lower extremity and a double atrophy, more marked on the right side. The only focal sign of brain tumor was an incomplete superior alternating type of paralysis. This was the only sign which at first led to the belief that the tumor was in the base of the pes pedunculi on the right side or in the middle fossa. Against the first supposition was the poorly marked paralysis of the extremities and the involvement of the fifth nerve on the right side. It seemed more probable that the tumor had begun in the medial portion of the middle fossa on the right side near the sella with a tendency to penetrate the sphenosphenoidal fissure. This would explain the unilateral syndrome which was present. The roentgen-ray examination showed that the sella was enlarged and flattened near its entrance, thus making for an extra sella tumor. There were absent the fatty dystrophies, the stupor, polyuria, glycosuria and the mental symptoms together with the homonomous hemianopsia which one sees in tumors of the sella. The patient was operated on, but died soon after.

The ninth observation was of a tumor of the sella with acromegaly associated with ankylosis of the spinal column. That this patient had developed a marked acromegaly was deduced from the history, from a measurement of the hands and face and from the characteristic appearance of these and of the feet. That a tumor of the hypophysis was present was suspected from the alterations in the sella and the clinoid processes and from a complete absence of disturbance of vision, which is explained by the fact that the sella was restricted above by an approximation of the clinoid processes thus allowing for an extension of the tumor downward and therefore away from the chiasm. The importance of this case lies in the fact that the acromegaly had developed many years after the onset of the arthritis deformans spinalis. The patient had root pains, lower motor neuron disturbances and reaction of degeneration in the right lower extremity and radiographic evidences of the vertebral involvement in the lumbar region with almost complete disappearance of the intervertebral disks and rarefaction of the bodies. The author after a review of the literature has failed to find a similar case. He believes that the pains in acromegaly, referred to by authors, are due in many cases to association of this disease with arthritis deformans spinalis. He also believes that the two conditions have the same etiology, namely, a dyscrasia glandulae.

The tenth case was a tumor of the clivi sella and hypophysis. The patient began to suffer from headaches, vomiting, dizziness and mild optic neuritis followed shortly afterward by focal symptoms consisting of paresis in the third, sixth and seventh cranial nerves, with slight increase in tendon reflexes. On the right side there was involvement of the third, sixth, seventh and fifth nerves, blurring of the outlines of the disks, diminution of the senses of taste and smell, cranial percussion, pain in the right occipital region and seven months later, mental dulness and somnolence. An examination of the brain at necropsy explained the presence of various symptoms. The pressure of the tumor on the right half of the pons and pedunculus explained the paralysis of the cranial nerves, including the oculomotor. Despite the intense pressure on the right half of the bulb, there were absent any symptoms of tenth, eleventh and twelfth cranial nerve disturbance. The fact that from the beginning there were focal signs pointing to the pons, would lead one to believe that the tumor began in the distal portion of the hypophysis, extending backward and upward on the clivus, and then finally invading the entire pons.

The eleventh case was a tumor of the right rolandic area. The headache was localized in the right temporoparietal region, there was dim vision, jacksonian attacks in the face and both extremities on the left side, loss of vibration sense in the left upper extremity, gradual paresis in the left face, upper and lower extremity, and pain on percussion over the right temporoparietal region. The irritative motor signs began by preference in the left upper extremity, and then became diffused through the left side. This left no doubt as to the site of the lesion. A tumor was diagnosed in the cortex cerebri in the prerolandic gyrus of the left (typographical error, should read right) side, which later extended to the postrolandic region. In this case there was absence of vomiting and dizziness and, despite the disturbance of vision, no changes in the fundi, symptoms which are characteristic of circumscribed tumors of the rolandic area. Operation confirmed the diagnosis. No necropsy examination was allowed.

The twelfth case was a tumor of the left prefrontal lobe. The patient's condition began with nocturnal headaches, vomiting, epileptiform seizures, papilledema (bilateral), increase of albumin in the spinal fluid, tachycardia, amnesia for words—this symptom being most marked for the language which he used least—a tendency to fall to the left and backward, mild and transitory disorientation, amblyopia and paresis of both sixth nerves and of the right third with exophthalmos most marked on the left side. On the left side there was anosmia, the head being turned toward the left, exaggeration of the patellar and pain on pressure of the eyeball. On the right side there was paresis of the fifth, seventh and twelfth nerves and of the extremities, hyporeflexia of the cornea, exaggerated tendo-achillis reflex and diminution of vibration sense in the toes, painful percussion of the head, diminution of vision and adiadokocinesis. It was easy to exclude a tumor in the rolandic area because of the absence of jacksonian seizures and dissociated paralyses; of the occipital lobe, because of the absence of hemianopsia and the presence of the amnesia, pain on percussion in the frontal region and the cerebellar gait. One might suspect the cerebellum because the cerebellar gait was present early in the disease, but the other symptoms excluded this possibility. By exclusion, the site of the tumor was localized in either the temporal or frontal lobe of the left side. There is always great difficulty in making a differential diagnosis between tumors in these positions. All the symptoms which this patient presented might accompany a left temporal tumor, if one admits that it began on the surfaces of the anterior portion of the temporal lobe. Against this possibility would be the diminution of vibratory sensibility and the rotation of the head toward the left. These symptoms, however, appeared late, and for a long time the exact localization of the tumor was not possible. The fact that pain on percussion was most marked over the frontal bone of the left side was a great aid. Against this symptom the absence of motor aphasia was of little value because many tumors of the left prefrontal lobe have been reported, even those which involve the third frontal and the insula, run through their course without this symptom. The absence of this sign, however, made for the localization of the tumor in the medial rather than in the lateral portion of the convexity of the lobe in question, which is an important point for the surgeon. To him it is not a matter of indifference as to whether the medial, orbital or convex portion of the frontal lobes are involved or whether, if the latter, the superior and medial frontal gyri are affected rather than the inferior. Durret has attempted to discriminate conditions in these localities, but the author is not prepared to accept them without reserve.

This case was also important because it illustrated the fact that psychic disturbances are not always present to a marked degree in frontal lobe tumors. The patient never suffered from the "Witzelsucht" of the German authors. Müller recently desired to have this symptom taken from the category of signs of frontal lobe tumor, and Stern has asserted that the presence of this condition depends on differences both local and individual and that the condition found is not always a euphoria. The psychic complex depends on the personality of the individual and, in the average case, consists merely of mental dulling and apathy. This is illustrated in the author's case, the patient being acutely interested in his condition to the last. However, the author agrees with Stern that usually in tumors of the frontal lobe the mental symptoms occur rather early and consist of loss of attention, apathy, memory disturbances and finally, when the tumor is exerting great pressure, mental dulling. The tumor discussed by the author was extramedullary. This type of tumor is more usually accompanied by mild mental disturbances, whereas, in subcortical tumors, the mental diturbances are profound and early. Stern recalls the fact that cerebellar tumors, which are in relation to the cerebrum as extramedullary, produce but rarely any marked mental disturbances excepting the terminal mental dulling. Many authors agree to this point, namely, that subcortical frontal tumors produce early and pronounced psychic disturbances, whereas, cortical tumors are not so apt to do so. Raymond thinks that this is due to the fact that the commissural and tangential fibers of association are destroyed. The paresis of the face and extremity of the right side, in the author's patient, is explained on the theory that a mild compression of the rolandic motor area subcortically had taken place. This also explains the absence of the irritative jacksonian seizures. The fact that the right patellar reflex was diminished was due probably to an irritation of the cerebellum on the opposite side by compression, which would also explain the mild hypotonia and the transitory adiadokocinesis. There were diminution of vibratory sense and paresthesia in the right extremities for the same reason, namely, compression of the postcentral gyrus and the lower parietal lobe. Interesting, too, in the author's case was the tendency to fall to the left and backward and the ataxic cerebellar gait which has been referred to, particularly by Bruns, as occurring in frontal and temporal lobe tumors. Bruns believes that the center of the musculature of the trunk is to be found in the prefrontal lobe which controls walking in erect posture. This center controls the automatic cerebellar centers for the maintenance of equilibrium. The author agrees with this conception, although he feels that it is not always due to pressure or injury of the frontocerebellar pathways, which have indirect connection with the central nuclei of the vermis of the opposite side. In his case, the tumor being on the left side, the right cerebellar hemisphere should have been the one involved. To explain the ataxia and the tendency to fall toward the left, one must believe that the left cerebellar hemisphere was affected indirectly by compression. The author refers to the symptom of rotation of the eyes and head to the right in his patient, and recalls that at necropsy it was found that the left second frontal convolution was found completely destroyed. He recalls that Bruns has found a center in this convolution in monkeys, which governed the movements of the head and eyes toward the opposite side. It would appear, therefore, that the foot of the left second frontal convolution governs the oculo-cephalo-gyric movements of the opposite side.

In tumors originating in the basal surface of the prefrontal lobe, the ocular muscles may become paralyzed and the first branch of the trigeminus

may become the site of pain caused by compression on the cavernous sinus where the oculomotor and ophthalmic branch are in close proximity. This would explain why his patient was unable to move the eyeballs in any direction except downward. The compression of the sinuses mentioned also explains the exophthalmos and the tenderness of the eyeballs. The author discusses what he calls amnesia verborum and explains that his patient tended to express himself in only one of the four languages which he spoke, namely, his native language, French. At necropsy it was found that the entire area of Broca was pressed on but not destroyed, and while the transitory dysarthria is well explained by pressure on the pars opercularis of the third frontal and of the foot of the precentral gyrus and possibly even of the left lenticular, nevertheless, so far as the amnesia verborum is concerned, we are obliged to say that this was due to distant compression on the temporal lobe, destruction of which the author believes is the cause of sensory aphasia. One is led to suppose that the faculty of recalling words heard cannot possibly be localized in any special zone as many believe, but is spread through the entire frontal and temporal lobes. The recalling of a word is a very delicate constituent of the comprehension of language. Therefore, even a mild disturbance will accentuate the loss of this function. The author has frequently observed in abscesses of the left temporal lobe, that the amnesia verborum is an early sign and that the sensory aphasic signs soon disappear after the abscess is evacuated, but the recalling of words is the last symptom to return, being difficult of reacquisition because it is a high, associative function. The same difficulty, namely, the difficulty of recalling, is seen in the senile psychoses and in neurasthenia cerebri. The author ends his article by referring to the great aid which the roentgen ray gave in this last case. The plates revealed a white area corresponding to the frontal lobe on the left side, probably due to a disappearance of the internal table of the skull.

American neurologists who do not know Mingazzini are referred to the many works on neuro-anatomy and pathology which this sterling worker has written.

OSNATO, New York.

POISONING BY HYDROCYANIC ACID GAS, WITH SPECIAL REFERENCE TO ITS EFFECTS ON THE BRAIN. SAMUEL W. LAMBERT, Neurol. Bull. **11**:93 (March) 1919.

The author gives a detailed description of a case of hydrocyanic acid gas poisoning, which, in view of its rarity, the meagerness of reports on the subject, and more particularly the excellent gross and microscopic pathologic studies of the brain lesions and the numerous illustrative plates, is well worth reviewing somewhat at length.

An Italian disinfector was found unconscious after having lain for twenty minutes in a room filled with hydrocyanic acid gas. The immediate symptoms noticed were: stertorous breathing, flushed skin, engorged veins of red color, contracted pupils, nonreacting to light; abdominal muscles firmly contracted, extremities rigid, temperature, 99; pulse, 120; respiration, 36. A few hours later the pulse came down to 70, the pupils alternately dilated and contracted, he perspired freely, there were involuntary defecation and micturition and frequent muscular twitchings. The urine showed a slight trace of albumin. Blood count revealed: 6,200,000 red blood cells, 15,100 white blood cells with 90 per cent. polymorphonuclears and hemoglobin 90 per cent.

During the following days the pulse remained slow most of the time, res-

piration in the twenties, temperature varied from 99 to 104, tonic convulsive seizures set in, a doubtful Babinski reflex appeared, sweating continued, there was difficulty in swallowing, the body was rigid; then the knee jerks disappeared, as did the Babinski reflex, tonoclonic convulsions appeared several times a day. After a few days the patient came out of his stupor, recognized people and carried out some commands, at which time a fine tremor and ataxia of his arms were discovered. He could see and hear, but there seemed to be an aphasia. Three days antemortem he grew more stupid, and died sixteen days after being poisoned. At first the spinal fluid showed 207 cells, of which 45 per cent. were polymorphonuclears and 55 per cent. lymphocytes; subsequently there were only 120 cells with 60 per cent. polymorphonuclears and 40 per cent. lymphocytes.

General necropsy revealed acute parenchymatous nephritis. The brain (reported on by Dr. F. Tilney): The meninges were adherent along the edges of the superior longitudinal sinus. On macrotonic section the entire white substance was much softer than usual; numerous small hemorrhagic areas were seen in the frontal and occipital lobes, the white substance of the centrum ovale and cerebellum. Microscopically, the cortical layers were found normal in architecture. The large pyramidal cells showed cloudy swelling. In the prefrontal area there was a decided gliosis. The Betz cells showed irregular outline, cloudy swelling and eccentric nuclei. The cerebellum had the most pronounced changes in the lateral lobes and vermis. The Purkinje cells disappeared almost entirely; there was marked atrophy and irregularity of the molecular and granular layers. There were numerous small hemorrhagic areas; many rod-shaped cells of unusual character were scattered profusely, seemingly independent of the vessels. There was no neuronophagia in the cerebellum as there was in the precentral area. The most marked changes were in the Purkinje cells.

An analysis of the symptoms shows the initial coma to have been due to the temporary paralytic effect of the toxin on the motor cell. The hypertonic condition is explained by the "irritative poisoning of the cerebellum with a continuance of the original disturbance of the frontal lobes," the contracted pupils by cortical irritation, the ataxia by the cerebellar lesion, the tonic convulsions by direct toxic effect on the motor cells of the cortical area, the aphasia by the degenerative condition of the motor cells in the speech area.

Aside from the fact that the case is very unusual, it is extremely interesting to the neurologist because of the complete study of the brain lesions. Most cases are either immediately fatal, allowing little time for brain changes to occur, or else recover without leaving much damage behind. Previous clinical reports of cases which survived several days seem to have given fairly analogous pictures and no doubt had similar pathologic brain lesions. It is interesting to note that the radical, cyanogen, besides exerting specific toxic effect on the hemase of the blood, thus not allowing the freeing of the oxygen from the hemoglobin, also seems to have a special toxic effect on the Purkinje cells of the cerebellum.

The microphotographs illustrate exceedingly well the hemorrhagic changes in the various parts of the brain cortex, cerebellum and centrum ovale, and a few normal sections from the temporal cortex serve to bring out the toxic changes in the large cells of the affected parts of the brain.

WECHSLER, New York.

Society Transactions

CINCINNATI NEUROLOGICAL SOCIETY

Regular Meeting, Sept. 25, 1919

FRANK W. LANGDON, M.D., *President*

A SPECIMEN OF BRAIN TUMOR. Presented by DR. CHARLES E. KIELY.

The patient, a boy, aged 19, was admitted to the Cincinnati General Hospital, Aug. 4, 1919. The chief complaint was convulsions, accompanied by screaming, which had first been observed in July of the same year. He also complained of diplopia. Examination showed normal fundi, slight ptosis, paresis of the superior rectus on the right, sluggishness of the right pupil to light and very slight weakness of the right peri-oral muscles. On looking to the left there was nystagmus with the fine component to the left. The result of vestibular tests was negative except for hypersensitiveness. The spinal fluid was under increased pressure, but laboratory reports were negative. The first diagnosis was disseminated sclerosis with hysteria, as many of the seizures were obviously functional and amounted merely to episodes of screaming.

In the next three weeks the patient became deaf, his whole right side weak and examination of the optic disks showed papilledema. Within another week he was lethargic and the papilledema had reached a point where the ophthalmologist advised decompression for the conservation of vision. The patient at no time had headache or vomited. Bradycardia appeared only a few hours before the operation. A sub-occiputal decompression was made by Dr. John A. Caldwell. An opening in the skull about one inch wide and extending an inch and a half to either side of the median line was made. On opening the dura the cerebellum herniated into the opening and no pulsation could be detected so that the object of the decompression was not attained. The patient died twenty-four hours later and an unskillful necropsy was performed, the brain being much lacerated and distorted. There was no neoplasm in the right cerebellopontine angle, but on account of the condition of the tissues, its point of origin could not be determined. A tag of the tumor was adherent to the right occipital lobe of the cerebrum. Paraffin sections showed it to be an endothelioma.

OPERATION FOR INJURY OF THE ULNAR NERVE. Discussed by DR. JOHN A. CALDWELL.

He presented two patients who had been operated on for injury of the ulnar nerve. Both were battle casualties. The first patient had been wounded in the Argonne in October, 1918, receiving a gunshot wound of the arm on the inner side just above the elbow. He had had no primary treatment other than débridement. Dr. Caldwell had first seen him in February, 1919, at which time the patient had paralysis and atrophy corresponding to an ulnar lesion. Deep sensation was retained and there was tingling on percussion of the nerve trunk, but cutaneous sensation was gone.

Operation in March by Dr. Dean Lewis revealed the ulnar nerve embedded in scar tissue. It was freed and surrounded by a layer of fat. At the present time there is considerable improvement, but there is still marked atrophy of the muscles of the hand supplied by the ulnar nerve. Until the past few weeks he has had daily massage and electrotherapy.

The second patient received a wound from a high explosive, Nov. 11, 1918, on the inner side of the arm above the elbow, completely severing the ulnar nerve. It was sutured the following day. When the patient was seen by Dr. Caldwell in August, 1919, there was evidence of complete division of the ulnar nerve. An incision extending 4 inches above and below the elbow was made over the nerve, and it was freed from surrounding scar tissue. Longitudinal incisions in the nerve disclosed that practically the entire cross section was infiltrated with scar tissue and consequently no conduction could be expected. To remove all of the infiltrated nerve required the sacrifice of 4 inches. The proximal segment of the cut nerve was loosened from its normal bed for about one third the distance up the arm and was passed through the deep fascia to a position just lateral to the median nerve. The distal segment was freed from its normal position and passed under the pronator teres, flexor carpi ulnaris and palmaris longus to a position just to the inner side of the median. With the ends of the nerve transplanted to the flexor side of the elbow, there remained a gap of but an inch and a half. This gap was closed by flexion of the arm to an acute angle, and the nerve was then sutured. The arm was held in this position for three weeks. The angle was then changed at intervals of two weeks until after six weeks all splints were removed. At present the ulnar nerve can be palpated as a tight cord on the flexor side of the elbow when the arm is two thirds extended. Of course, no return of function is seen at this time.

Attention was called to a phenomenon that has frequently been noticed in peripheral nerve injuries. In the first case, which had simply an incarcerated nerve, there was atrophy of extreme degree of the hypothenar eminence, the adductor of the thumb and the interossei; while in the second case, in which there had been complete interruption, there was some atrophy, but it was not nearly so marked.

Book Reviews

DIE NERVOSITÄT ALS PROBLEM DES MODERNEN MENSCHEN: Ein Beitrag zur Psychologischen Weltbetrachtung. HERBERT OCZERET, Med. Prakt. (Zürich), Zurich: 1918. Druck und Verlag: Art. Institut Orell Füssli. Pp. 95. Price, 3.80 francs.

Apparently this interesting little monograph is meant more for the laity than for the medical profession. It is in part an essay, an oration, a harangue and a disquisition. Some paragraphs are suggestive of the slang words "barnstormer" and "spellbinder." But the author appeals to one as the free-lance always does. He praises Freud, with reservations; commends Adler, but stabs him in vulnerable places; and lauds Jung, but criticizes some of his dicta.

The first part is largely a bitter impeachment of the general medical profession for its ignorance of, and indifference to, the nature of "nervousness," which the author treats as more or less of an entity.

Chapter two contains an intelligent discussion of the views of Freud, Adler and Jung. Without complete acceptance of Freud's views, the author gives him great credit for formulating a really mental treatment for mental disorders and carrying us beyond the stage of virtual electrodes and asafetida. He also commends Freud's moral courage in "rubbing under our noses" one of the weightiest social problems of the times, but intimates that an intemperate enthusiast was required for such an undertaking.

Having asserted that "nervousness" has enormously increased since 1750, the author refers it to changes in the mental attitude and emotional life of the common people, and especially to the development of self consciousness, individuality and freedom of personal feeling and action; also to the concomitant freer, more complicated and more emotional relations between men and women.

Some space is devoted to an estimate of the social value of the neurotic. A neurosis is born of nonvictorious conflict, but does not necessarily imply low value of the person (Minderwertigkeit). It is due to (1) complexity of problem, (2) complexity of the modern psyche or (3) ignorance of the individual ego and his lack of appreciation of necessary living conditions. On these assumptions, the author thinks that many a neurotic, like any delicately constructed and finely adjusted machine, may be one of the most valuable productive items in our modern cosmos—if properly understood and handled. He gives Bismarck as an example, a known neurotic, but a most effective and valuable citizen. However, he does not show that Bismarck was understood and wisely handled, but that the man himself had the intellect and stamina to manage himself.

Nearly half of the ninety pages of text are on the rearing of children; one case is related in detail. As a plea for closer attention and better understanding, the matter is good but there is little in the way of practical instruction.

The problem of the modern woman is given twenty-six pages, apparently because "woman as such is a nascent or rising (emporsteigend) class." The problem of the modern man is accorded but fourteen pages. Is it because

his song is now *diminuendo?* In the section on woman the author gives advice to both husband and wife in the form of a long letter to each containing unexceptionable, fatherly advice. The ideas are good and well expressed, the advice is sound and the decisions rational. But will either man or woman abide by them?

DIE NERVOSITÄT BEI DEN JUDEN: Ein Beitrag zur Rassenpsychiatrie für Aerzte und gebildete Laien. Dr. Med. Rafael Becker, Assistenzarzt der Kantonalen Heil-und-Pflege-Anstalt in Königsfelden bei Brugg, Zürich, 1919. Druck und Verlag: Art. Institut Orell Füssli. Pp. 31. Price, 2 francs.

That functional nervous disorders and mental disorders are more frequent in Jews is conceded, this being supported by the best authorities as well as by the author's personal experience. But that one may conclude therefrom that the Jews have a predisposition to such disorders, that is, "have something in them which allows them to become nervously or mentally sick more frequently than others," is warmly contested by the author.

There is presented a rather detailed consideration of the incidence of the common forms of mental disease and disorder, including epilepsy and a summary of various views on the subject, but the principal thesis of this essay is that the nervousness of Jews is due to no degeneracy but to former and present social conditions. It is a bit startling to read that "in these modern times the Jews suffer much greater torments than their forbears in the middle ages and earlier times, the times of torturing, the rack and *autos da fé,*" but the author's argument on this is interesting. He says that in those days the Jew had his faith and was a joyful, even exalted martyr, whereas now he has the feeling of one who is looked down on, with no spiritual recompense.

Evidently the essay was written for Jewish readers, but it is interesting, well written and not unfair.

WAR NEUROSES AND SHELL SHOCK. Frederick W. Mott, M.D., LL.D., F.R.S., F.R.C.P., Brevet Lieut.-Col., R.A.M.C. (T.), Senior Neurologist to the Maudsley Neurological Clearing Hospital; Consulting Physician, Charing Cross Hospital; Director of the Pathological Laboratory of the L. C. C. Asylums; Corresponding Member of the Neurological and Psychiatrical Societies of Paris. With Preface by the Rt. Hon. Christopher Addison, M.P., Minister of Reconstruction. Pp. 348. London, Henry Frowde, Oxford University Press, Hodder and Stoughton, Warwick Square, E. C., 1919.

So far as we know, this is quite the most extensive work on war neuroses that has appeared. Apparently it aims to cover the subject in a complete and systematic way and does treat of nearly all the multifarious phases of war neuroses more fully than has yet been done in a single book. The author is an able neurologist quite exceptionally trained in the physiology and organic pathology of the nervous system and peculiarly skilled in its microscopic examination, but at the opening of the war he was obviously unprepared by his previous experience for effective and fruitful study of the psychoneuroses. Indeed, he frankly avows his earlier (quite gross) errors in the explanation of shell shock, but he still retains in this book a great deal of his earlier organic interpretation. He also introduces a considerable disquisition on surgical shock and shock in general, all of which has little to do with war neuroses, but it is very interesting and informing. It seems a bit of a pity

that this excellent monograph should not be brought really up to date by eliminating most of the author's earlier speculation produced when he was earnestly groping for the light—which he subsequently found. But elsewhere, also, the volume shows evidence of being in considerable part built up by assembling a lot of previous disjointed papers and notes.

The book begins with a classification of the effects of high explosives (*sic*) on the central nervous system: (1) the immediately fatal cases; (2) cases of wounds and injuries not fatal, and (3) affections of the central nervous system without visible external injury.

Although the author recognizes that "the psychogenic factor is by far the most frequent and important" one in the genesis of psychoneuroses and although the term "shell shock" has been officially banished from the British army, he still preserves the term, with his own personal definition. We think this unfortunate, as general use of the term has already done much harm in and out of the army. Further, the best of men disagree as to what shell shock is or ought or ought not to be. Certainly there is no general agreement with Colonel Mott when he says:

> . . . "Shell shock" is a useful term if it is limited to cases where there is definite evidence of a shell or bomb bursting near enough to knock the man down, or blow him up in the air and cause a temporary loss of consciousness."

We also doubt that all competent observers will agree to "the fact that a man who has suffered from true shell shock is not fit to return to general service for six months at least."

The book is based on an enormous personal experience and probably nowhere can be found a more complete array of cases of all kinds or a more reliable report on the microscopic changes in the nervous system to be found after severe concussion or physical commotion.

Sixteen pages are devoted to dreams and many interesting and instructive cases are cited. While the ideas of Freud and others are considered, the author seems to be scarcely at home in this field and takes the sayings of literary philosophers such as Lucretius and Shakespeare as seriously as the work of psychologists. To be sure, the exact value of the work of the latter investigators has not been determined.

Following the section on dreams is a chapter of seventy pages on war psychoneuroses, although these disorders receive more or less attention in other parts of the book. Few neurologists will seriously dissent from the opinion that "the psychopathology of war consists fundamentally in the exaggeration and preservation of instinctive defense reactions incidental to normal physiological conditions, viz., protective pain, fatigue and emotion." The various functional motor disorders are considered in some detail and under the head, "Examination of the Patient," are catalogued all the principal findings: motor, sensory, special sense, cardiovascular, gastro-intestinal, etc. The part on differential diagnosis is weak and the better portions are taken bodily from the French. On page 139 it is stated that "hysterical hemiplegia with contracture or hysterical hemicontracture develops all at once." Any one familiar with hysteria knows that this may sometimes be true but frequently is not.

The author's attitude regarding Babinski's ideas on physiopathic or reflex disorders is not exactly clear, but he seems to give them a somewhat half-hearted endorsement.

There are a couple of pages on "The Mental Conflict in Relation to War Psychoneuroses." Many readers will consider them quite inadequate. Per-

haps they were written carelessly, for they seem to say that such conflicts are limited to officers, which is, of course, perfectly absurd.

Thirty pages are devoted to gas poisoning, principally by carbon monoxid. A large part of this matter is old, but it is exceptionally good and of value in civil as well as in military conditions. Other interesting and instructive things in the book are a consideration of windage and its effects, voltaic vertigo, shock in relation to memory (including musical memory) and hysterical speech defects.

The section on treatment (30 pages) is far from complete but well balanced and reasonable. It will be of great assistance to the general practitioner and contains hints of value to the expert. The consideration of war psychoses is very sketchy but may suffice for those not especially interested in the subject.

The bibliography is decidedly inadequate, the index excellent; the illustrations (eighty-one in number, of which three are in colors) are very good indeed, and printing and binding are entirely satisfactory.

It is a good book though it has some pretty obvious defects. It is quite the best that the general practitioner can get and one that no general neurologist or psychiatrist can quite afford to be without.

PSYCHOSES OF THE WAR, INCLUDING NEURASTHENIA AND SHELL SHOCK. H. C. Marr, M.D., Lieut.-Col., R. A. M. C. (Temp.); Fellow of the Royal Faculty of Physicians and Surgeons, Glasgow; Neurological Consultant to the Scottish Command; H. M. Commissioner of Control for Scotland; Formerly Specialist in Nervous Diseases to the Troops in Malta (1915-1916); Physician Superintendent of Glasgow District Mental Hospital, Woodlee, Lenzie; Referee, Under the Workmen's Compensation Act for Dumbartonshire; and Mackintosh Lecturer in Psychological Medicine, St. Mungo's College, Glasgow, etc. Pp. 328. London, Henry Frowde, Oxford University Press; Hodder and Stoughton, Warwick Square, E. C., 1919.

In reading this work for review a sympathetic attitude becomes more and more difficult because, as one proceeds, evidence accumulates of a fine opportunity lost and of much valuable clinical material gone to waste. Having made this somewhat caustic declaration, the reviewer feels obligated at least to indicate his justification.

Discontent begins with the Introduction, which is a loosely written mixture of anatomy, physiology, pathology and clinical psychiatry, in which the known is not well discriminated from the hypothetical and relative values are not in accord with modern medicine. Under "Means of Observation of Mental Phenomena," the author stresses almost exclusively facial expression, a sort of sublimated physiognomy, properly and decently buried years ago.

In Chapter 2 the author indicates that shell shock and neurasthenia are the same thing, a subhead being "Neurasthenia (Shell Shock)," and states that "hysteria is in reality a symptom complex of neurasthenia." Having agreed with Charcot that hysteria is a mental condition, in the next sentence it is "a functional affection of the cortical, bulbo-spinal and sympathetic neurones." Nowhere does one find a definition of neurasthenia, but the author's division of it into (1) simple; (2) hysterical, and (3) organic is sufficiently inclusive. Under "Neurasthenia of Toxic Origin" he alludes to a frequent expression of soldiers exposed to shell explosion in a confined place and draws certain conclusions therefrom. The soldiers say the explosion has "taken the

guts out of them" and therefore Dr. Marr concludes that "these results can only be ascribed to the effect of concussion on the abdominal sympathetic nervous system." An illustration on page 52 bears the legend, "Facial Expression of Fear in Hysterical Depression (Toxic Neurasthenia)," and on page 69 we read: *"Observation.—Hysterical neurasthenia from bacillary infection; mental confusion and emotionalism. Exciting cause; shell explosion. Recovered after use of autogenous vaccines."* Did the shell explosion cause the bacillary infection? The account of the case does not answer the question. Nor is it made clear whether the vaccines or the sinusoidal baths (also used) cured the patient or whether he wearied of both and just naturally got well.

Observation 6, in the section on malingering, is labeled, "Simulation of deaf-mutism in neurasthenia," and the text says the patient "really suffered from hysterical deaf-mutism." Does this indicate loose classification or careless writing or slovenly thinking?

Again, one is struck by the satisfied conclusiveness with which the author introduces a diagram of brain centers and association tracts to make clear the phenomena of sleep and dreams. But it must be conceded that to diagram is much easier than lucidly to explain.

Ordinary phobias are classed as "insane obsessions." Janet's psychasthenia is made to carry some long since discarded ideas of the older psychiatry as well as forced to include "mental rumination" in the category of "forced spasmodic movements." On pages 132 and 133 patients who present obvious constitutional inferiority, not to say high grade imbecility, are cited as psychasthenics.

The book abounds with interesting illustrative cases, but essential details are frequently lacking and the reader must make his own interpretations; those of the author seldom are very helpful.

In approaching the subject of treatment of the psychopathies such expressions as "undermined the nervous system and the inherent constitution of the neurones," and "all external influences which irritate the wounded mind," would lead one to expect some glittering generalities; and they are there. But the author is sufficiently explicit in recommending Easton's syrup, the effluve from a Wimshurst machine, and sinusoidal baths—especially the last. To be sure he is not clear as to whether the quite remarkable effects of the baths are due to suggestion or because they "act directly on the circulatory system by causing a relaxation of high arterial tension." Nor does he even hint how relaxation of high arterial tension really acts on the circulatory system nor how, this action having been attained, the psychopathy is affected by it.

One hundred and three pages are devoted to mental deficiency, toxic psychoses and organic psychoses, though of course no particular relation of these affections to the war is shown, except some statistical statements as to their relative frequency. In these pages the reviewer has been able to find nothing new of value but quite a bit that generally has been discarded. Here, too, is found considerable looseness of classification and vagueness of outline, not to mention some careless use of the English language which contributes its mote to the prevalent lack of clarity.

The book closes with thirty-four pages of an elaborate scheme of "mental case-taking." The scheme contains some excellent and practical ideas and should be consulted by any one formulating a plan of record for an institution or for some special purpose.

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